Assessment Of Nutritional Status In Cirrhosis And Its Impact On Complications

DISSERTATION SUBMITTED FOR

DM MEDICAL GASTROENTEROLOGY

BRANCH- IV

AUGUST 2015

THE TAMILNADU Dr.M.G.R.MEDICAL UNIVERSITY, CHENNAI,

TAMILNADU.

CERTIFICATE

This is to certify that this dissertation entitled **"Assessment Of Nutritional Status In Cirrhosis And Its Impact On Complications**" submitted by **Dr. Vishnu Abishek R** to the Faculty of Medical Gastroenterology, the Tamilnadu Dr.MGR Medical University, Guindy, Chennai-600032, in partial fulfilment of the requirement for the award of DM Degree, Branch IV (Medical Gastroenterology) is a bonafide work carried out by him under my direct supervision and guidance, during the academic year 2012 to 2015.

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DECLARATION

I **Dr. Vishnu Abishek R** declare that I carried out this work on "**Assessment Of Nutritional Status In Cirrhosis And Its Impact On Complications**"at the Department of Medical Gastroenterology, Govt.Peripheral Hospital and Kilpauk Medical College. I also declare that this bonafide work or a part of this work was not submitted by me or any other for any award, degree, diploma to any university, board either in India or abroad.

This is submitted to the Tamilnadu Dr.M.G.R. Medical University, Chennai in partial fulfilment of the rules and regulation for the D.M. Degree examination in Medical Gastroenterology.

Govt. Kilpauk Medical College **Dr.Vishnu Abishek R**

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CERTIFICATE OF APPROVAL

The Institutional Ethical Committee of Govt. Kilpauk Medical College, Chennai reviewed and discussed the application for approval "Assessment of Nutritional Status in Cirrhosis and it's Impact on Complications"- For Project Work-submitted by Dr.Vishnu Abishek.R, DM Post Graduate, Department of Medical Gastroentology, Govt Kilpauk Medical College, Chennai-10.

The Proposal is APPROVED.

The Institutional Ethical Committee expects to be informed about the progress of the study any Adverse Drug Reaction Occurring in the Course of the study any change in the protocol and patient information /informed consent and asks to be provided a copy of the final report.

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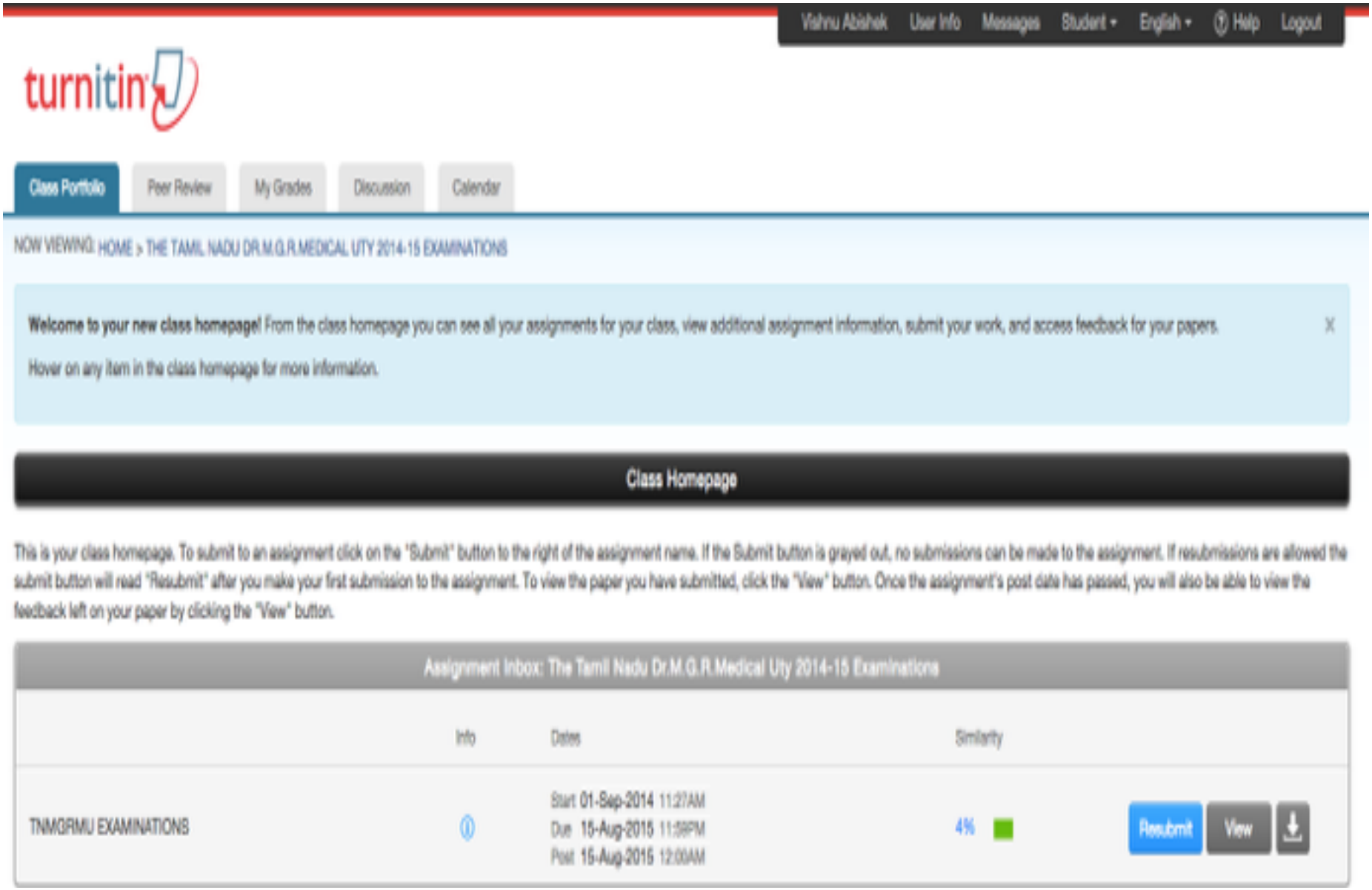
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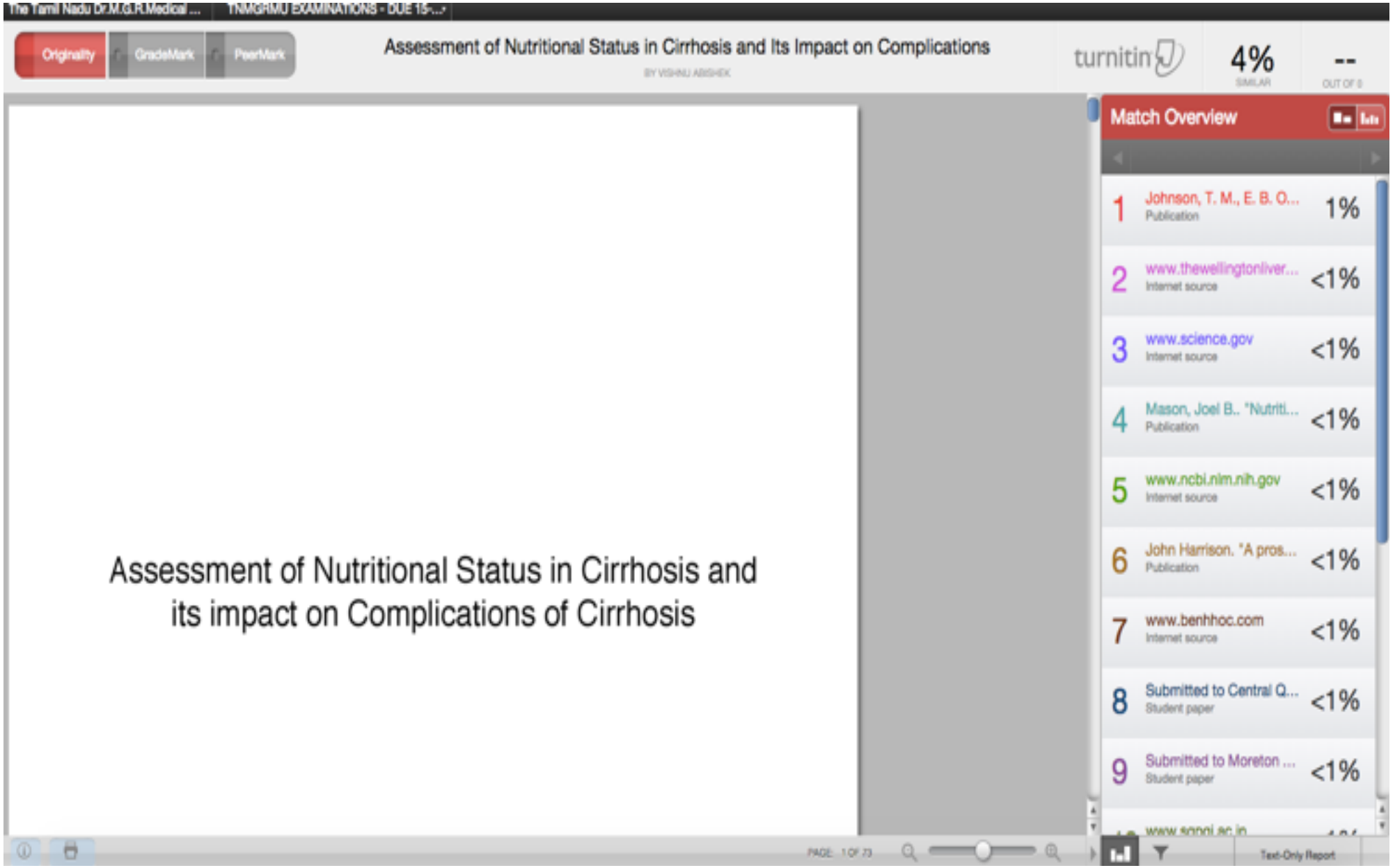
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Keywords:

Cirrhosis, Alcohol, Malnourishment, Subjective Global Assessment Score, Child Turcotte Pugh Score, Anthropometry, Albumin, BodyMass Index, Triceps Skin Fold Thickness, Mid Arm Muscle Circumference, Hand Grip Dynamometry, Bio Impedance Analyses, Complications, Spontaneous Bacterial Peritonitis, Hepatic Encephalopathy, Medical Nutritional Therapy

Abbreviation used:

CTP: Child Turcotte Pugh
SGA: Subjective Global Assessment
BMI: Body Mass Index
TSFT: Triceps Skin Fold Thickness
MAC: Mid Arm Circumference
MAMC: Mid Arm Muscle Circumference
HGS: Hand Grip Strength
HE: Hepatic Encephalopathy
SBP: Spontaneous Bacterial Peritonitis
ROC: Receiver Operator Characteristic
SE: Standard Error of Mean
CLD: Chronic Liver Disease

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Introduction

Cirrhosis is a chronic parenchymal liver disease, defined histologically as a diffuse hepatic process characterised by fibrosis and the conversion of normal liver architecture into structurally abnormal nodules. In India there was estimated 188,575 cirrhosis related death in 2010[1] accounting for 18.3% of global burden of cirrhosis related deaths. The loss of productivity and scion economic burden on family of the patients is unaccounted. The cause of the morbidity in cirrhotics is multifactorial and one may be attributable to malnourishment. Protein Calorie Malnourishment in Cirrhosis is synonymous with protein and fat nutrient deficit. The Quality of Life is abysmal with end stage cirrhosis per se and it is further worsened by complication like Infection especially SBP, GI Bleed , Encephalopathy, Renal Failure and other complications. Bedside parameters to detect malnourishment early in the course of the disease may be useful in providing medical nutritional therapy and reducing the morbidity and mortality in cirrhosis.

Review of Literature

Effect of Malnourishment on the course of Cirrhosis:

Major nutrient deficient is protein in cirrhotics [2], though trace elements and vitamins deficiency also are quiet common. Body composition is also altered in CLD wherein Extracellular Fluid volume expands and Intracellular Fluid volume contracts [3]. Prevalence of malnutrition in cirrhosis ranges from 50% to 90%. Men are more prone for malnourishment, an effect hypothesised due to low testosterone in cirrhosis. Alcoholic Cirrhosis are more undernourished than post necrotic cirrhosis patients. Malnourishment negatively affects immune function, respiratory effort, muscle mass, wound healing. Death rate is high in overly malnourished. Ascites and HRS positively correlates with Protein Energy Malnourishment. The causes of malnutrition is in table 1. The severity of malnourishment increases the event of complications due to cirrhosis.

Table 1

Aetiology: Malnourishment in Cirrhosis[5]	
Pathosiosiology	**Causes**
Low dietary intake	Dyspepsia Ascites Encephalopathy Gastroparesis Restrictive diet (low sodium, low protein, fluid restriction) Dysgeusia (zinc deficiency) Alcohol intake Socioeconomic status Increase in leptin
Hyper catabolism	Increase in sympathetic activity Insulin resistance Increased REE Decreased Respiratory Quotient (decreased aerobic glycolysis and lipid peroxidation)
Malabsorption of nutrients	Cholestasis SIBO
Defect in hepatic storage of nutrients **Enteral Protein Loss**	

Figure 1 shows the increase in mortality with increasing severity of poor nourishment.

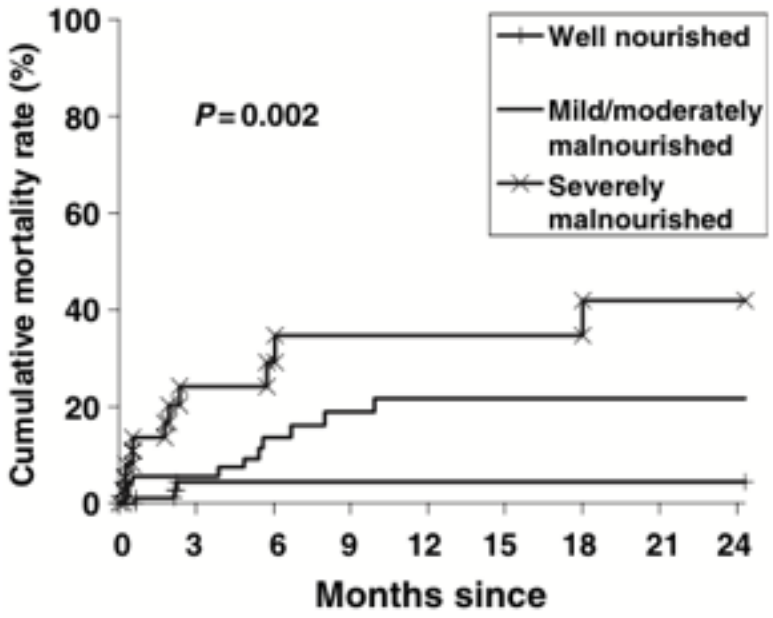


Figure 1:

Kaplan Meir plot showing cumulative mortality rate in cirrhosis as per SGA score[6]

Physiological Effects of Protein Energy Malnourishment in Body:

1) *Effect on GIT*: Structural and functional atrophy of small intestine, decreased conjugated bile acids in bile , reduction in gastric and pancreatic secretion, proliferation of anaerobic bacteria in small bowel, malabsorption of other nutrients like carbohydrates, fat and vitamins.

2) *Effect on Cardiovascular system*: Structural and functional loss of cardiac muscle mass, impaired myocardial performance.

3) *Effect on Immune system*: The most affected by PEM, T cell function is uniformly impaired, B cell Antibody repose is only variable affected. Hence they are prone for infection

4) *Effect on Respiratory system:* Decreased vital capacity and respiratory effort

5) *Effect on Endocrine system*: Low Insulin, Somatomedin levels, Raised Cortisol levels contributes to hyper catabolism. Reverse T3 is raised,with low T3 and T4 cause reverse T3 syndrome and sick euthyroid syndrome. Primary Gonadal Dysfunction is common due to PEM[29].

Cirrhosis and dysregulated metabolism

Liver is a metabolic factory for proteins, carbohydrates and lipids alike. Key site is sinusoids.

Glucose Metabolism

In healthy individuals, liver maintains plasma glucose levels by gluconeogenesis and gycogenolysis. This homeostasis is maintained by relative concentration pf Insulin and Glucagon. Raised Insulin to Glucagon ration inhibits gluconeogenesis and promotes glycogen synthesis, glycolysis and fatty acid synthesis. Cirrhotics have increased frequency of hyperglycaemia and relative hyperinsulinemia[7]. The hyperglycaemia is attributed to decreased glycolysis and reduced glycogen storage in liver and muscle, which is compensated for by increased gluconeogenesis. Also, increased FFA reduce glucose uptake by muscle. Glucagon and catecholamine levels are raised which is also contributory towards hyperglycaemia. In summary there is decreased glycogen synthesis, increased gluconeogenesis and increased insulin resistance.

Protein Metabolism

Liver in health has a synthetic function of albumin, globulin and fibrinogen synthesis. Ammonia production by deamination of amino acids and subsequent conversion to urea occurs exclusive in liver. Gluconeogenic amino acids are alanine and glutamine, ketogenic amino acids are leucine and lysine, producing free fatty acids. Amino acids in liver take part in production of glutathione and creatine, an important energy source in skeletal muscles. In cirrhotics, gluconeogenic amino acids glutamine and alanine are provided by proteolysis in skeletal muscle, which also contributes to the muscle

wasting. In summary, there is protein catabolism, decreased urea synthesis, and increased Branched Chain Amino Acid utilisation for ketogenesis)

Lipid Metabolism

Liver is the hub of cholesterol degradation and excretion and production of lipoproteins. Hypertriglyceridemia is the most common lipid abnormality in cirrhosis, due to increased fatty acid production and decreased beta oxidation. LCAT production by liver is impaired, causing a reduction in plasma cholesteryl ester levels.

Abnormal cell membrane lipids and abnormal erythrocytes like echinocytes are an effect of chronic dyslipoproteinemia.[8] In summary, there is increased lipolysis, fatty acid oxidation and ketogenesis.

Micronutrients

Fat soluble vitamins are deficient because of the decreased synthesis of carrier and transport proteins by liver. Vitamin A deficiency in particular increases the risk of Hepatocellular carcinoma in Cirrhosis. Vitamin D and Vitamin E deficiencies are seen in 90 % and 50% of cirrhosis patients respectively.[9] Clinically they manifest as osteoporosis, osteopenia, haemolytic anaemia, increased platelet aggregation. Water soluble vitamins thiamine, pyridoxine, vitamin B12 deficiencies are common. Thiamine and Folate deficit occurs much rapidly in cirrhosis. Manifestations are neuropsychiatric. Vitamin B12 may be falsely elevated because the

laboratory assay includes metabolically inactive Cobalamin analogues (Transcobalamin I and III).[10]

Trace Elements: Zinc deficiency deficiency occurs due to enteral and urinary Zn loss, and it causes precipitation of Hepatic Encephalopathy by affecting urea cycle enzymes and glutamine synthesis.

Other manifestations are altered taste and smell, immune dysfunction, depressed mental state.[11] Homocysteine, cysteine, copper levels are raised. Choline is deficient manifesting as impaired verbal and visual memory, magnesium deficiency manifesting diarrhoea, depression and muscle weakness, and both of them causes worsening of hepatic disease. Manganese excretion is impaired in End Stage Liver Disease, causing its deposition in basal ganglia, altered central dopamine metabolism and structural changes in astrocytes. Peripheral type BZD receptors is increased by Manganese contributing to the pathogenesis of HE.

Body composition

In healthy people, the body composition is as follows.[25]

i) Extra cellular lean mass 36%

ii) Fat mass 28%

iii) Muscle mass 22%

iv) Visceral mass 7%

v) Blood cells/bone cells 7%.

End stage liver disease patients have increase in ECF volume and decrease in ICF volume. As in any illness state, instead of over utilising fat, Protein is lost.

Protein loss is reflected in low Fat Free Muscle mass (FFM) in cirrhotics, wherein there is greater loss of muscle FFM than non muscle organ FFM. As a result Non Muscle FFM in vital organs like heart, brain and kidney raise the metabolic demand and Resting Energy Expenditure in Cirrhosis. Hence Cirrhosis is a hyper metabolic state, which in reality is a not a favourable state as it results in increased wear and tear, more prone for malnutrition and poor prognosis[12]

Poly Unsaturated Fatty Acids:

In Cirrhosis PUFA is deficient, as it is synthesised in liver. However PUFA supplementation cirrhosis worsened the malnourishment. Hence PUFA deficiency might actually be a adaptive response to counter the decrease in ATP.

Resting Energy Expenditure (REE):

REE is defined as the energy expenditure in a awake and idle state in the in between meals period. Normally in Adults, 1 Kcal/Kg/Hour of Energy Liver demands 19% of REE of the body. REE is calculated in clinical practice using Harris Benedict Equation in clinical practice. However the gold

standard is Indirect Calorimetry, which measures heat produced by oxidation[58].

Men = 66 + (13.7 × W) + (5 × H) − (6.8 × A)

Women = 665 + (9.6 × W) + (1.8 × H) − (4.7 × A)

Cirrhosis being a hyper metabolic state, increases the REE. After PEM sites in, the REE is reduced. Hyper metabolism is seen only in 70%. Any complication in cirrhosis like infection, GI bleed, raises the stress factor, hence the Total Energy Expenditure (TEE) increases. Adjusted body weight is used to measure REE as actual body weight underestimates energy requirements.

TEE = REE x Stress factor

Stress factor depends on the clinical condition ; example, Stress factor in peritonitis is 1.2, and in severe infection its is 1.3

Nutrition Assessment Parameters

Measuring the weight is grossly inaccurate in nutritional status assessment in ESLD, because of fluid overload. Hence BMI can be normal in patients with Protein Calorie Malnourishment, and it cannot be used a s tool to assess nourishment status. Subjective Global Assessment (SGA) questionnaire used as a nutrition assessment tool in cancer patients and paediatric patients. It has been found to be useful in Cirrhosis as well. SGA A is no malnourishment, SGA B is mild and SGA C is severe malnourishment respectively.

Objective assessment parameters include Triceps Skin Fold Thickness, Mid Arm Muscle Circumference, Hand grip dynamometry, Bio impedance analysis, DEXA, Body cell mass.

Nutritional Intake assessment

It as always difficult to accurately quantify the nutritional intake in patients, howe ever there are tools available for the purpose. 24 hour recall, food frequency questionnaire, calorie count and food dairy are some of them. None of them have been validated.[13]

Subjective Global Assessment Scale, a score which is based on 4 patient subjective parameter of weight loss, GI symptoms, functional capacity, Food intake and also on physician's assessment of muscle wasting, oedema, and ascites, is modified by Hasse et al for use in cirrhosis patients[51]. SGA scale is used to accurately assess malnutrition, in cancer patients and in paediatrics, now also used in cirrhosis patients. SGA scale is in figure 2.

figure 2

Subjective Global Assessment

Name:
Date:

Medical History			A	B	C
WEIGHT	Usual weight.....	Current weight.....			
Wt change past 6 months	Amount weight loss.....	% weight loss.....			
0-<5% loss			*		
5-10% loss				*	
>10% loss					*
Weight change past 2 weeks		Amount.....			
No change; normal weight			*		
Increase to within 5%			*		
Increase (1 level above)			*	*	
No change, but below usual wt				*	
Increase to within 5-10%				*	
Decrease					*
DIETARY INTAKE					
No change; adequate			*		
No change; inadequate				*	
Change		**Duration of change**.....			
Suboptimal diet				*	
Full liquid				*	
Hypocaloric liquid					*
Starvation					*
Intake borderline; increasing			*		
Intake borderline; decreasing				*	
Intake poor; no change				*	*
Intake poor; increasing				*	
Intake poor; decreasing					*
GASTROINTESTINAL SYMPTOMS					
	Frequency (never, daily, no. of times/week)	Duration (<2wk, >2wk)			
Nausea	.....	.....			
Vomiting	.....	.....			
Diarrhoea	.....	.....			
Anorexia	.....	.....			
None; intermittent			*		
Some (daily >2 week)				*	
All (daily >2 week)					*
FUNCTIONAL CAPACITY					
No dysfunction		Duration of change	*		
Difficulty with ambulation/normal activities				*	
Bed/chair-ridden					*
Change past 2 week					
Improved			*		
No change				*	
Regressed					*

Physical examination	A	B	C
SUBCUTANEOUS FAT			
Under the eyes	Slightly bulging area		Hollowed look, depression, dark circles
Triceps	Large space between fingers		Very little space between fingers, or fingers touch
Biceps	Large space between fingers		Very little space between fingers, or fingers touch
MUSCLE WASTING			
Temple	Well-defined muscle/flat	Slight depression	Hollowing, depression
Clavicle	Not visible in Males; may be visible but not prominent in females	Some protrusion; may not be all the way along	Protruding/prominent bone
Shoulder	Rounded	No square look; acromion process may protrude slightly	Square look; bones prominent
Scapula/ribs	Bones not prominent; no significant depressions	Mild depressions or bone may show slightly; not all areas	Bones prominent; significant depressions
Quadriceps	Well rounded; no depressions	Mild depression	Depression; thin
Calf	Well developed		Thin; no muscle definition
Knee	Bones not prominent		Bones prominent
Interosseous muscle between thumb and forefinger	Muscle protrudes; could be flat in females		Flat or depressed area
OEDEMA (related to malnutrition)	No sign	Mild to moderate	Severe
ASCITES (related to malnutrition)	No sign	Mild to moderate	Severe
OVERALL SGA RATING	**A**	**B**	**C**

Adapted from: Detsky et al., 1994[8]; Baxter Healthcare Corporation, 1993; McCann, 1996 (Ferguson, Bauer, Banks, Capra, 1996)©

Table 2:

Subjective Global Assessment Scale	
A	Well Nourished
B	MIld to Moderate Malnourishment
C	Severe Malnourishment

Biochemical Parameters

Pre-albumin (Transthyretin), Albumin, Transferrin, Retinol Binding Protein (RBP) are synthesised by the liver and their levels are less in Advanced liver disease. These parameters can be used in prognostication, but they cannot be used in accurate assessment of nutritional status, as they are more characterised as negative acute phase reactants and indicate underlying inflammation and disease process, rather than the synthetic function of liver. [14]

Creatinine Height Index: amount of 24 hour urine creatinine, corrected for patient's height accurately assesses the skeletal muscle mass. Values that are less than 20% of normative values are suggestive of severe Protein Calorie Malnourishment. Abnormal kidney function, milk ingestion, toured administration can erroneously affect the values. The usefulness of this test in Cirrhosis is not validated.

Anthropometry

1) Height 2) Weight 3) BMI 4) Triceps Skin fold Thickness 5) Mid Arm Circumference 6) Mid Arm Muscle Circumference (MAMC)

Cirrhosis patients are volume overloaded, hence mere weight or BMI may not bring out the malnourishment to clinician's view. BMI may be normal (18 .5 to 23.5) in the setting of ascites and oedema, though FFM is lost. Campillo et al[27] found that cut off for BMI in identifying malnourishment in cirrhosis if

below 22 in the absence of ascites, below 23 in the presence of moderate ascites, and below 25 Kg/m2 in the setting of massive ascites. This cut off has not been validated by others, and varies in different racial background. Hence an approximate estimate of whole body fat mass is measured by the subcutaneous fat thickness, which is approximately 50% of body's adipose tissue. Triceps skin fold thickness (TSFT) or sub scapular fold thickness is measured as a surrogate marker of subcutaneous fat thickness[15]. Skin fold thickness is measured using specially designed callipers, like Harpenden's calliper. Hypoalbuminemia and skin oedema may affect the measurement in decompensated cirrhosis.

Mid Arm Muscle Circumference (MAMC) measures the skeletal muscle mass approximately. However confounding factors of gender and different humerus diameter has to be taken in to account and its not a accurate predictor of malnutrition when taken as a lone parameter[6.] MAMC is measured from Mid Arm Circumference (MAC) and Triceps Skin fold thickness (TSFT) using the following formula.

MAMC = MAC - (0.314 x TSFT)

MAMC and TSFT Values less than 5th percentile for a population is considered abnormal and used in diagnosis of malnutrition. Normative values of MAC and Skin Fold Thickness as per western standards are in Table 2 & 3, (courtesy: NHANES I and II)

Table 3

Mid Arm Circumference Median value (cm)		
Men	25-30 years of age	53
	45-50 years of age	55
	65-70 years of age	48
Women	25-30 years of age	30
	45-50 years of age	32
	65-70 years of age	35

Table 4

Sum of triceps and sub scapular fold thickness (mm), median value	
Men 45-50 years	24
Women 45-50 years	37

Assessment of Body Composition

The following are the techniques used in Body Compartment Analysis [17,18,19,20]

1) Proportion of lean mass and fat mass is calculated using the following methods, a) Underwater (hydrostatic) weighing b) Air displacement plethysmography

2) DEXA scan: Absolute fat mass, lean mass and bone density is measured by Dual energy x-ray absorptiometry. It is less accurate for lean mass in DCLD because of increase in ECF water.

3) Total body potassium is used to calculate Body cell mass

4) Isotopically labeled water and NaBr dilution calculates TBW, ICW, ECW

5) In vivo neutron activation analysis is used to measure Total body protein, absolute Fat Mass, absolute Lean Mass. Neutrons are used to bombard the tissue elements and gamma camera are used for picking up signal from elements, thereby measuring total body protein, calcium ,electrolytes etc.

6) CT, MRI: regional Lean mass and Fat mass are assessed.

The above tests are accurate and effective methods of measuring body compartment but they expensive, not widely available, and hence not in routine use outside research purpose.

7) Bio Impedance Analysis (BIA) : It is a cost effective method of measuring FFM, body cell mass (BCM), and total body water (TBW). The principle involved is electricity in the limits of 800 micro amp, when passed through the body is met with resistance to flow because of fat and bone minerals, whereas tissues rich electrolyte laden water conducts the electricity. This makes accurate assessment of FFM, fat percentage, Body Cell Mass more cost effective and accurate.

REE and phase angle are additional parameters measured by BIA.Impedance consists of electric resistance and reactance.

Phase angle (PA) is the tangent value of the ratio of reactance versus electric resistance. There exists a correlation between PA values and body cell mass, and cell membrane integrity. Cirrhosis causes disruption in cell membrane integrity and thereby a low phase angle. Satish et al[45] study in Bengaluru showed that the phase angle in normal healthy subjects were7.32 $\pm$ 1.7^{o}. Peres et al[47] showed that phase angle less than 5.18^{o} caused an increase in relative risk increase of 2.5 for death in cirrhosis. However in Decompensated Cirrhosis, assessment of body water and fat percentage is inaccurate because of volume redistribution in third space.[21]

Functional Assessment of Protein Calorie Status:

There are 2 methods for functional assessment of protein calorie, by measuring muscle strength.

Hand Grip Dynamometry and Respiratory Muscle Strength are the parameters, and the third one Delayed Hypersensitivity Skin testing is a non specific way of implying protein malnutrition as PEM affects immune calls and Cell Medicated Immunity, resulting in decreased sensitivity of skin testing. It has several confounding variables and hence is grossly inaccurate.

Hand Grip Dynamometry measured using a hand held Dynamometer- digital or analogue- in units of Kilogram per Force, is an excellent tool in

determining depleted fat free mass in cirrhosis. It is paid and cost effective, but requires a little alert and co-operative patient.[22]

Respiratory Muscle strength is assessed using a spirometer. Maximum sustained inspiratory and expiratory force are measured. This has several confounding variable affecting the respiratory effort. Hence neither an easy or reliable method.[23]

Morbidity in Cirrhosis

Cirrhosis is prognostically classified based on Child Turcotte Pugh Score in to CTP A, CTP B and CTP C. clinically there are 4 stages of Cirrhosis[57]

Stage 1: Compensated, no ascites, no varies

Stage 2: Compensated, No ascites, but varices present, no bleeding

Stage 3: Decompensated with Ascites, with or without oesophageal varies

Stage 4: Decompensated with Variceal Bleed with or without Ascites.

Malnourishment in advanced cirrhosis increases the risk of complications in cirrhosis. Abbott et al. investigated the relationship between the CTP classification and nutritional indicators and found that advanced CTP classification was associated with diminished muscle status and greater early post-operative morbidity after liver transplantation. Casafont et al studies the

influence of malnourishment in the incidence of Spontaneous Bacterial Peritonitis in rats.[26] 100% of malnourished rats showed evidence of bacterial translocation, resulting in SBP. The incidence of Hepatic Encephalopathy if 25% in undernourished cirrhotics.[28] Nutritional deficiency adversely affects post operative period outcome in Liver Transplantation. A state of hyper catabolism, decreased glycogen synthesis and anaerobic glycolysis causes pro inflammatory cytokine raise and subsequent SIRS and Muti organ failure in post transplantation period.

Nutritional Supplementation in Cirrhosis:

Energy estimation

ASPEN guidelines[31] state that Compensated cirrhosis patients require 25-35 kcal/kg/day, and malnourished patients require 30-40 kcal.kg/day. Generally fat rich diet is given for PEM, as its provides highest calorie count per gram of supplementation. Lipid provides 20-40% of caloric needs, otherwise small frequent meal is recommended with evening time carbohydrate rich snack to prevent prolonged starvation which in turns predisposes to muscle protein catabolism. Protein restricted diet is no longer recommended in cirrhosis as Cordoba et al showed deterioration of Hepatic Encephalopathy on protein restriction and subsequent muscle proteolysis.[30] ASPEN guidelines also

suggest energy intake based on REE and stress factor. Stable cirrhotics with malnutrition is advised a total energy intake of REE x 1.2 to 1.4 , critically ill patients who are at risk of refeeding syndrome need to be started on a diet with 15 to 20 kcal/kg/day and gradually increasing to 25-30 kcal/kg/day on maintenance phase. A critically ill obese patient require calorie replacement based on calculations by Mifflin–St Jeor equation.

Protein requirement

To prevent muscle proteolysis and promote gluconeogenesis, 1.0 to 1.5 g/kg of protein is recommended. Transient Protein restriction to 0.6 to 0.8 g/kg/ day is recommended in acute exacerbation of HE. Vegetable protein is preferred over animal proteins as it provides a rich supply of Branched Chain Amino Acids (BCAA), decreases gastric transit time, increases intra luminal pH. A daily intake of 30-40 gram of vegetable protein is ideal.[32]

Fluid Intake:

Fluid is restricted to less than 1.5 L/day only in the setting of ascites with hyponatremia. Otherwise 30-40 ml/kg/day is allowed. However in clinical practice fluid consumption is individualised to suit the patient based on intake output calculation.

Carbohydrate and fat:

A range of 25% to 30% of total caloric needs is provided by lipids. Excess of lipids, more than 6 g/kg/day of glucose is not recommended. However a carbohydrate rich evening snack is recommended, as it helps in raising the total body protein levels irrespective of the stage of cirrhosis.[33]

Salt restriction:

< 2 g/day salt is advised to overcome fluid retention in DCLD.

Weight loss in Volume overload:

In Cirrhosis decompensated with ascites, standard of care is salt restriction to <2g/day and diuretics, under weight loss monitoring, to ensure that patients loses 0.5 kg/day, or a1k/day in the presence of edema. However, a hospitalised patient, in need of additional calorie requirement, when subjected to rapid weight loss measures like large volume paracentesis and diuresis, will lose carbohydrate and protein from body. This will further aggravate the malnutrition.[36]

Nutritional support:

Enteral support is preferred over parenteral nutritional support. Enteral support includes providing 1.5 kcal/ml of fluids via naso-enteric or naso-

gastric tube. Enteral supplement of 100 ml must contain 100 kcal of carbohydrates, 4 g of protein, 3.5 mol of sodium.

Routine use of BCAA rich hepatic formula feeds are not recommended. Calorie dense , low volume feeds are better tolerated by advance liver disease patients.[31] PEG tube placement in DCLD is not advised in the setting of ascites or varices as infection and bleeding in GIT is common. Parenteral nutrition is advised only if nutrition deficit could not be bridged by oral and enteral route. Strict glucose monitoring is warranted, and if hyperglycaemia is present, then glucose is limited to 2-3 g/kg/day, lipids to ≤1 g/kg/day. Cyclical Rotation of regimen is recommended to avoid cholestasis due to TPN and concentrated solution is used to avoid volume overload.[34] Sepsis and TPN catheter displacement are quiet common . Rotation must be in the form reduction in glucose load, lipid load and caloric content when LFT is abnormal due to TPN. Manganese and copper is limited in the setting of cholestasis.

Branched Chain Amino Acids:

Aromatic amino acids like phenyl alanine, tryptophan and tyrosine are raised in cirrhosis patients, while BCAA like leucine, valine, isoleucine levels are low. Serotonin precursor tryptophan crosses blood brain barrier and worsens

HE. BCAA will cause increased cerebral perfusion and decreases tryptophan crossing BBB by competing for Amino Acid transport. BCAA detoxify ammonia and promote muscle glutamine synthesis. Leucine stimulates hepatic protein synthesis by promoting hepatic growth factor secretion from stellate cells of liver. Nocturnal supplementation of BCAA adds to nitrogen balance and albumin synthesis, while day time BCAA supplies only calorie, not protein.[35] Though initial studies about BCAA supplements were not encouraging, subsequent large double blinded RCT by Marchesnini et al[37] favoured the long term administration of BCAA which arrested the hepatic failure progression. Nakaya et al study showed that Cerebral function improves positively with BCAA supplements, so does serum albumin levels and cell metabolism[38]. Oral granular form is more palatable and ideal for long term supplementation[39].

Anti-oxidant supplements

Oxidative stress is high is cirrhosis, with increase in pro inflammatory cytokines and reduction in the body stores of selenium, zinc and vitamin E.

Vitamin E supplementation has shown to reverse the inflammation and fibrosis in NASH[40], but it has no mortality or morbidity benefit in end stage liver cirrhosis[41].

N-Acetyl Cysteine (NAC is a glutathione prodrug. Glutathione crosses BBB and provides into oxidant function. Its role in improvement of Hepatic Encephalopathy is yet to established[42], but it has proven its efficacy in Hepato renal syndrome type 1 [43].

Vitamin Supplementation

Water soluble vitamins and fat soluble vitamins need to be replaced. Vitamin D hydroxylation in liver is affected, hence Calcidiol (25 OH Vitamin D) production is hindered. So it can be supplemented as oral Calcidiol (25-OH Vitamin D) 25-50 microgram capsules. Cirrhosis patients showing improvement with B complex vitamin supplements was detected way back in 1940s by Arthur J patter Jr.[44.]

Vitamin K is supplemented during variceal bleed. Thiamine, Folate, Vitamin B12 deficiency occurs faster in cirrhosis.

Prebiotic and Probiotic Supplements

Prebiotic promotes the growth of beneficial bacteria in intestine, while probiotics are the beneficial bacteria given in live form, synbiotics are a combination of both. Increase in lactic acid producing bacteria in GIT causes reduction in intra luminal pH which prevents pathogenic bacteria from producing ammonia, thereby reversing HE.[46]

Obesity and cirrhosis:

NAFLD patients developing cirrhosis, need urgent weight loss in compensated liver disease stage. Once they decompensate weight loss is beneficial, but they are also protein deficient, hence individualised nutritional therapy is warranted, as adequate protein supplement is needed[49] . Obesity with Diabetes needs a weight reduction of atleast 5-10% to alleviate insulin resistance. Rapid weight loss if not advised as it is a risk factor for decompensation[50].

Hepatogenous Diabetes:

Cirrhosis is a Insulin Resistant state. Impaired glucose tolerance and Diabetes can develop in a cirrhosis patient and cirrhosis is considered the likely aetiology for Diabetes if the Patient is a cirrhotic for more than half a decade, does not have metabolic syndrome, hemochromatosis or family history of Diabetes.[49] Ferritin levels are high in these patients.

Conclusion:

Metabolic dysregulation and Malnourishment in cirrhosis is a common entity, ant it negatively impacts the course of complication and mortality in Cirrhosis. BMI may under diagnose malnutrition. Specific biochemical , anthropometric parameters, Hand Grip Dynamometry, Bio impedance analysis, DEXA, Subjective Global Assessment score can identify malnutrition in apparently well nourished cirrhotics. Early identification can be helpful in providing medical nutritional therapy in cirrhosis, thereby reducing the morbidity and mortality.

Aims and objectives

Primary **:**

To asses the nutritional status in cirrhosis patients by anthropometry, biochemical markers, muscle strength assessment, Bio impedance analysis and subjective global assessment and to compare efficacy of each in identifying malnutrition and to assess the severity of malnourishment with the severity of liver disease.

Secondary **:**

To correlate nutritional status of the cirrhotic patients with the occurrence of cirrhosis related complications.

Materials and Methods

Study Design: Prospective, observational, cross sectional study

Study centre: Department Of Digestive Health and Diseases, Government Peripheral Hospital, Anna nagar, Kilpauk Medical College, Chennai.

Period of Study : November 2013 to February 2015

Study population: In patients and Outpatients in Department of Digestive Health and Disease, Government Peripheral Hospital, Anna Nagar, Chennai.

Inclusion criteria: Cases of cirrhosis diagnosed by clinical, biochemical and imaging parameters, age more than 18 years

Exclusion criteria: Hepatic encephalopathy stage 2 or more

Financial Assistance: Nil

Reference Group: Healthy volunteers accompanying patients

Methodology:

Baseline demographic values were recorded. Complications of cirrhosis if present were noted, like Spontaneous Bacterial Peritonitis and Hepatic Encephalopathy. Biochemical parameters like LFT, Serum Proteins, RFT were collected, CTP score calculated, and Anthropometric measurements were documented. (Height, weight, BMI).

a) Mid arm circumference was measured with inch tape at a midpoint between accordion process and olecranon in the relaxed non dominant arm.
b) Triceps skin fold thickness measured with callipers similar to the standard callipers, with a reading close to 0.1 mm
c) MAMC: Mid arm muscle circumference is calculated by using the formula MAMC = MAC in cm - (TSFT in mm x 0.314)
d) Hand grip strength was be assessed with Camry digital hand grip dynamometer. Average of three readings taken at a interval of 5 minutes was used as final value expressed in Kg/F.
e) Omron Karada Scan HBF-375 Body Fat Analyser was used for Bio Impedance Analyses. Bioelectrical impedance analyser was used to analyse body composition (skeletal muscle, Visceral fat and sub cutaneous fat in percentage). It also gave Resting Energy Expenditure, but that was not included in the study.

f) Subjective Global Assessment score was calculated as per Hasse's modified questionnaire. All patients were classified in to SGA A, SGA B, or SGA C based on the score. SGA 'A' is well nourished, 'B' is mild to moderately malnourished, 'C' is severely malnourished. These parameters were compared with each other, with CTP score and the complications occurring in these patients at the index assessment. All the parameters were compared with the values obtained from the reference population of healthy volunteers visiting the hospital as patient bystanders.

Statistics

Descriptive data was analysed using SPSS statistics software version 22. All quantitative data were expressed in Mean $\pm$ 2SE. ANOVA and the chi-square test were used to compare the standard error of mean. $P < 0.05$ was considered significant.

Results

Demographic data:

87 patients with cirrhosis were included in the study. 79 were male (90.8%) and 8 were female patients (9.2%) . In 72.41% (n=63) of the cases, alcohol was the cause for the cirrhosis. Chronic Viral Hepatitis was

the cause in 13.79% (n=12), 3 patients (3.4%) had Wilson's Disease, 4 patients (4.59%) had NAFLD, Cryptogenic cirrhosis in 5 patients (5.74%).

Figure 4

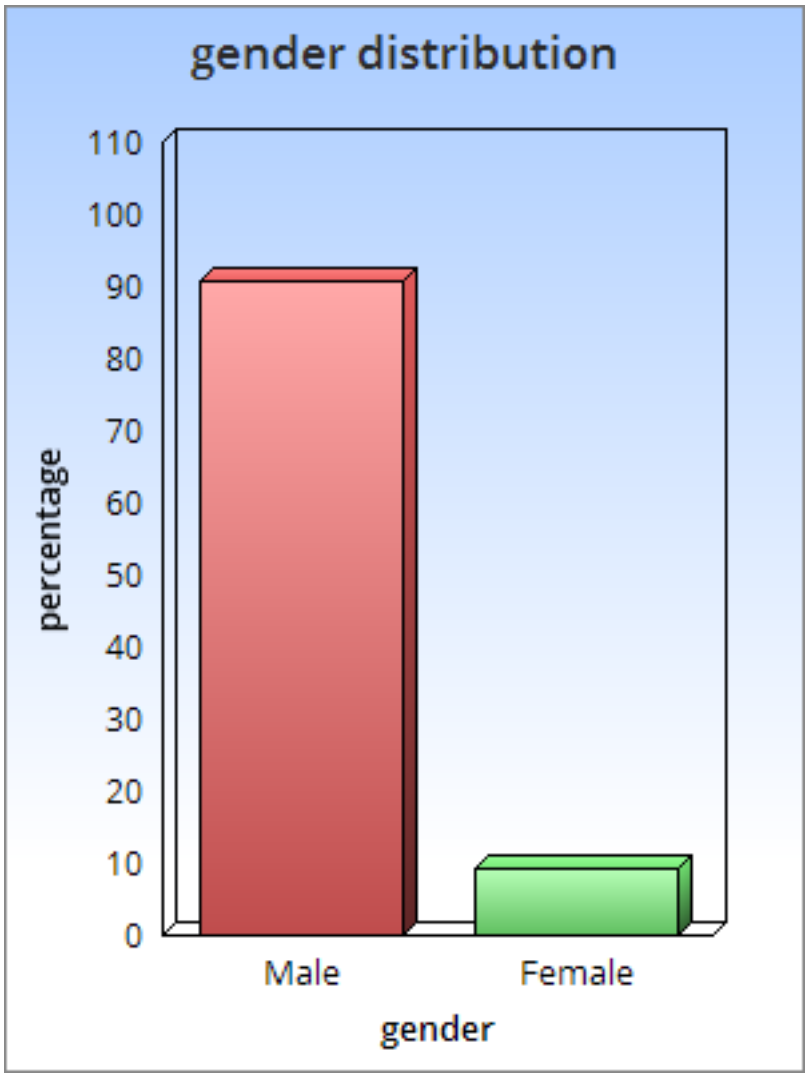
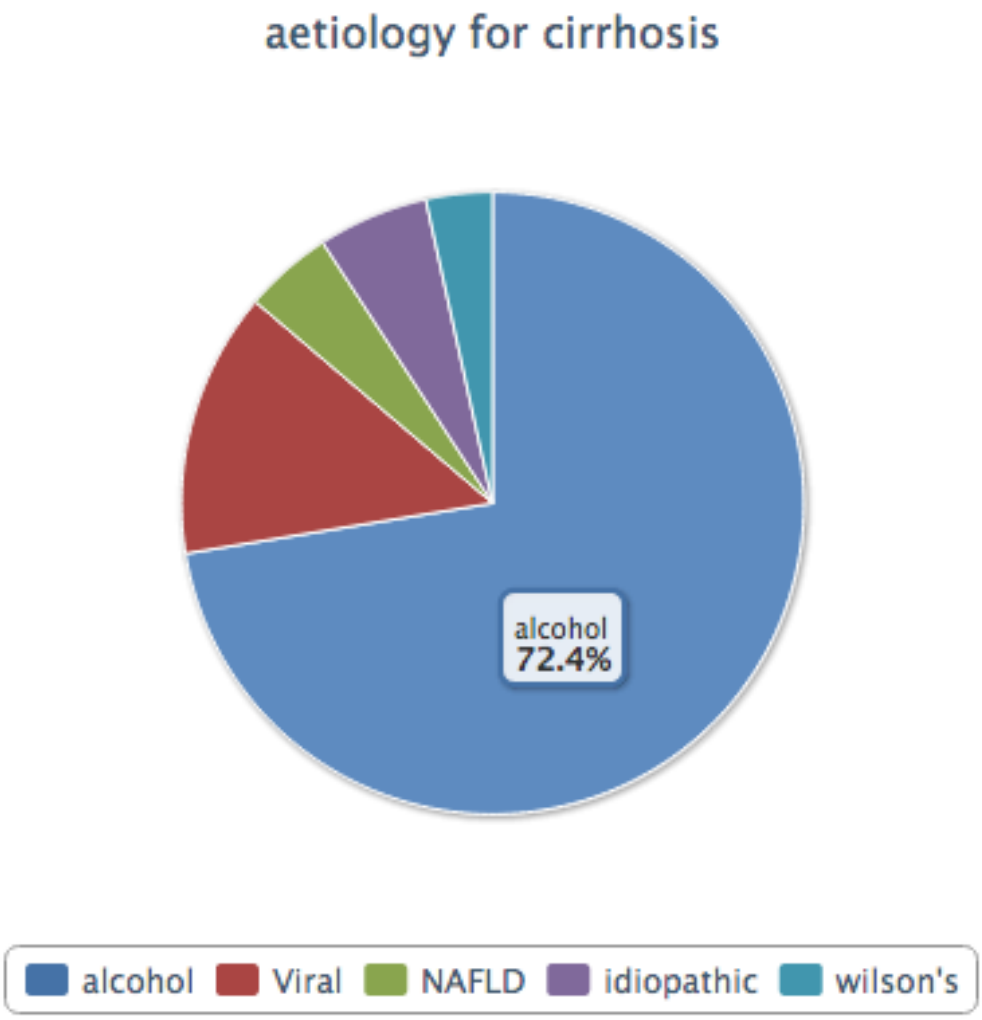


Figure 5



Diagnosis of Cirrhosis was made within a year's duration in 50.57% of patients (n=44), in another 24.13% the diagnosis was made after 1 year of disease duration and within 2 years at the time of assessment, and another 20.68% having cirrhosis for more than 2 years but less than 5 years duration. In total 95.38 % of patients were diagnosed less tan 5 years within the date of assessment.

62.06 % of the study population were in age group 4th and 5th decade age wise. outliers in the extremes of age were negligible, hence the parameters used to assess nutritional status was applicable to the entire study population.

Figure 6

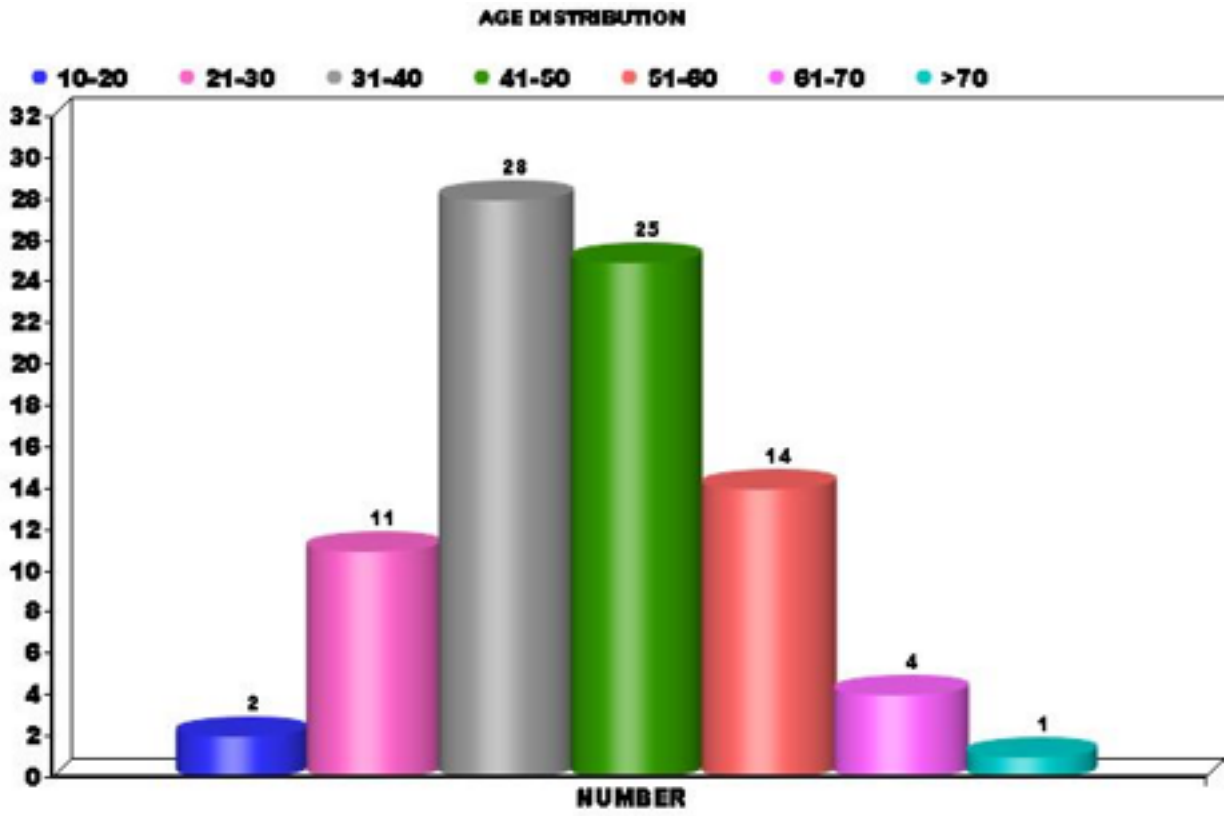


Table 5

Severity of Cirrhosis in study population: CTP classification	
CTP A	18
CTP B	27
CTP C	42

20.6% of the study group was in Child Turcotte Pugh Class (CTP) A. 48.7% of the study group was CTP class C.

80.4% of the study population had ascites

Table 6:

Body Mass Index of the Study population (kg/m^2)	
CTP A	22.91
CTP B	23.34
CTP C	22.9

The mean BMI in each CTP class showed no significant difference. This is because of the volume overloaded state attributable to ascites and oedema in 80.4% of our study group. Weight and BMI were in normal range even though they were malnourished with obvious muscle wasting. Hence BMI did not categorise malnourishment in our study

The Subjective Global Assessment Score was used and patients were classified in to SGA A,B,C, and accordingly 19 patients were in SGA A, 49 in SGA B, 19 in SGC C.

SGA group A is well nourished, hence cumulatively 68 patients(78.16%) were malnourished. On comparing the malnourishment diagnosed by SGA score with CTP score, there was a significant difference, with increased incidence of malnutrition with increasing severity of liver disease.

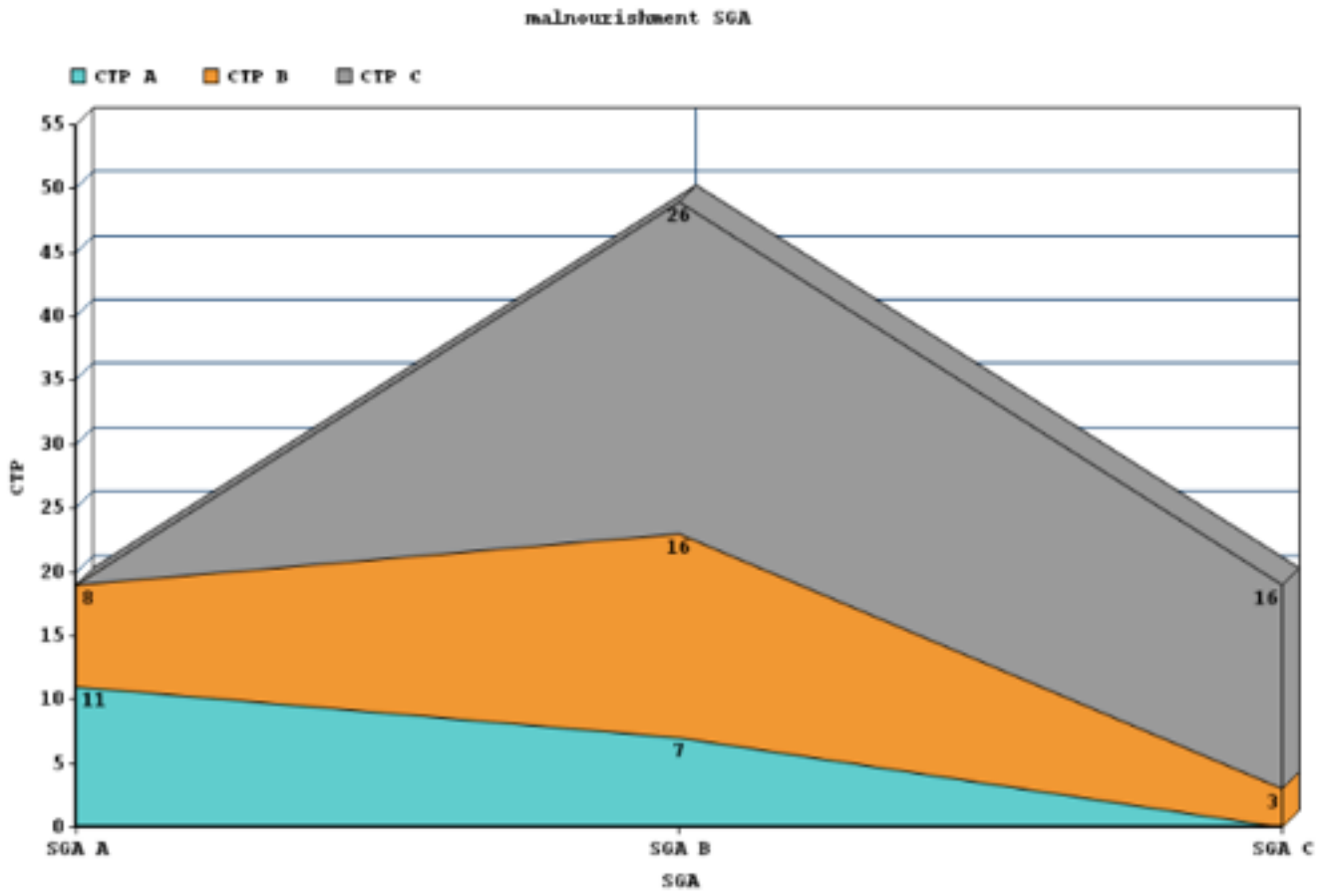
80.4% of the study population had ascites. Of the 17 patients who had no ascites, the prevalence of mil to moderate malnourishment (SGA - B) was 29.4% (n= 5/17).

According to SGA score for diagnosing malnutrition, even 38.8% of the patients in early stage Cirrhosis like CTP A were malnourished, mild to moderate. 100% of patients in CTP C were malnourished. Severe malnourishment was high in CTP C.

Table 7: Significance of difference in malnourishment between CTP class A,B and C

SGA	**A (19)**	**B &C (68)**	**Chisquare**	**P value**
CTP A	11	7		
CTP B	8	19	28.96	0.0001
CTP C	0	42		

Figure 7: SGA vs CTP



The Anthropometric data and Hand grip strength were measured and documented separately for male and females and tabulated against SGA group A, B and C, to obtain the significance of difference between well nourished and malnourished (table 8 and 9). Values were significantly low in Malnourished patients compared to well nourished, and it was even low in well nourished cirrhotics compared to normal reference population.

This difference was significant only in male population which comprised of 90.8% of the study group, and was not significant in female population with a too low sample size.

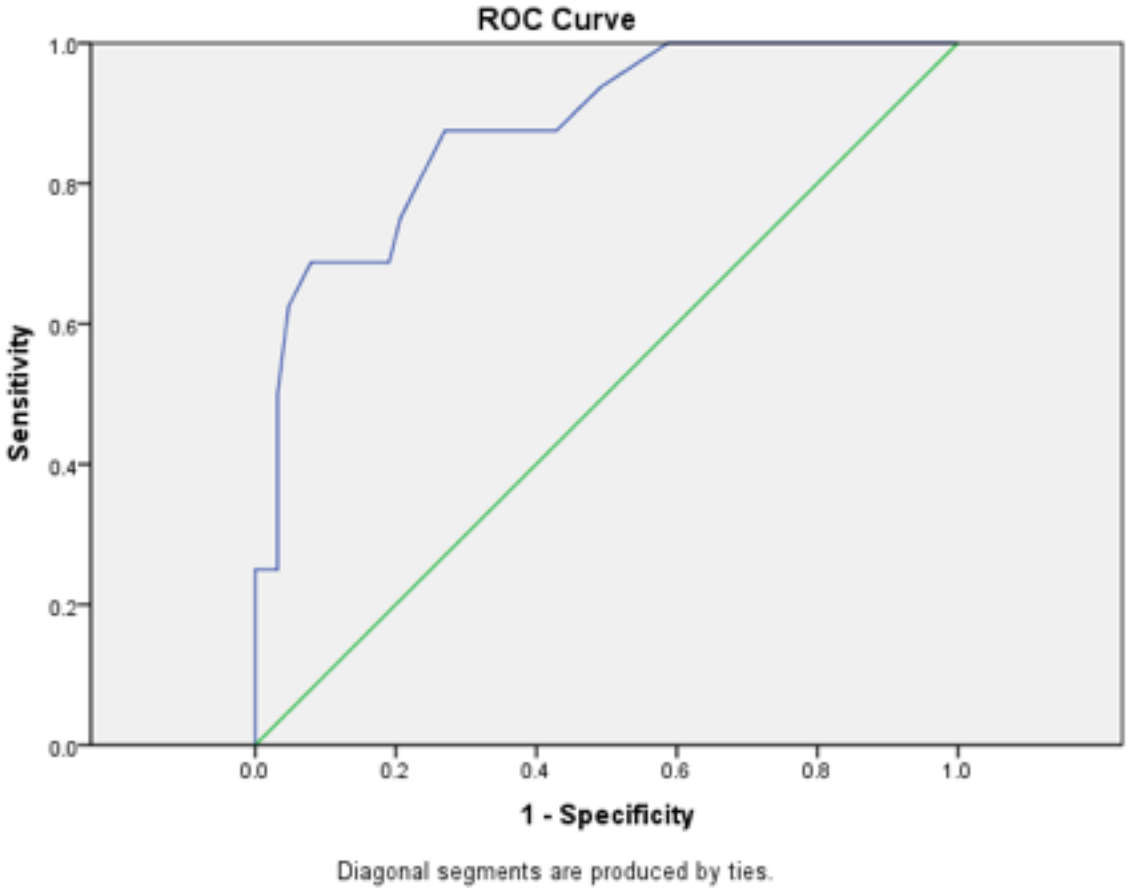
Table 8: Triceps skin Fold Thickness, Mid Arm Circumference, Mid Arm Muscle Circumference, Hand Grip Strength (mean) for male patients

SGA	TSFT (mm)	MAC (cm)	MAMC (cm)	Hand Grip Strength (Kg/F)
SGA A	12.37	28	24.19	31.87
SGA B	9.13	23.38	20.51	22.59
SGA C	6.57	21.13	19.06	17.6
healthy volunteers	15.6	31.2	25.24	38.7
p value	**0.0001 (F -21.98)**	**0.0001 (F - 21.06)**	**0.0001 (F -15.07)**	**0.0001 (F - 21.41)**

Table 9: TSFT, MAC, MAMC, Hand grip strength in females (mean)

SGA	TSFT (mm)	MAC (cm)	MAMC (cm)	Hand Grip Strength (Kg/F)
SGA A	12	25.83	22.06	22.07
SGA B	8.4	20.02	17.56	12.73
SGA C	-	-	-	-
healthy volunteers	21	28.5	23.36	25.56
p value	0.18	0.09	0.1	

Figure 9: ROC for Mid Arm Circumference: Male

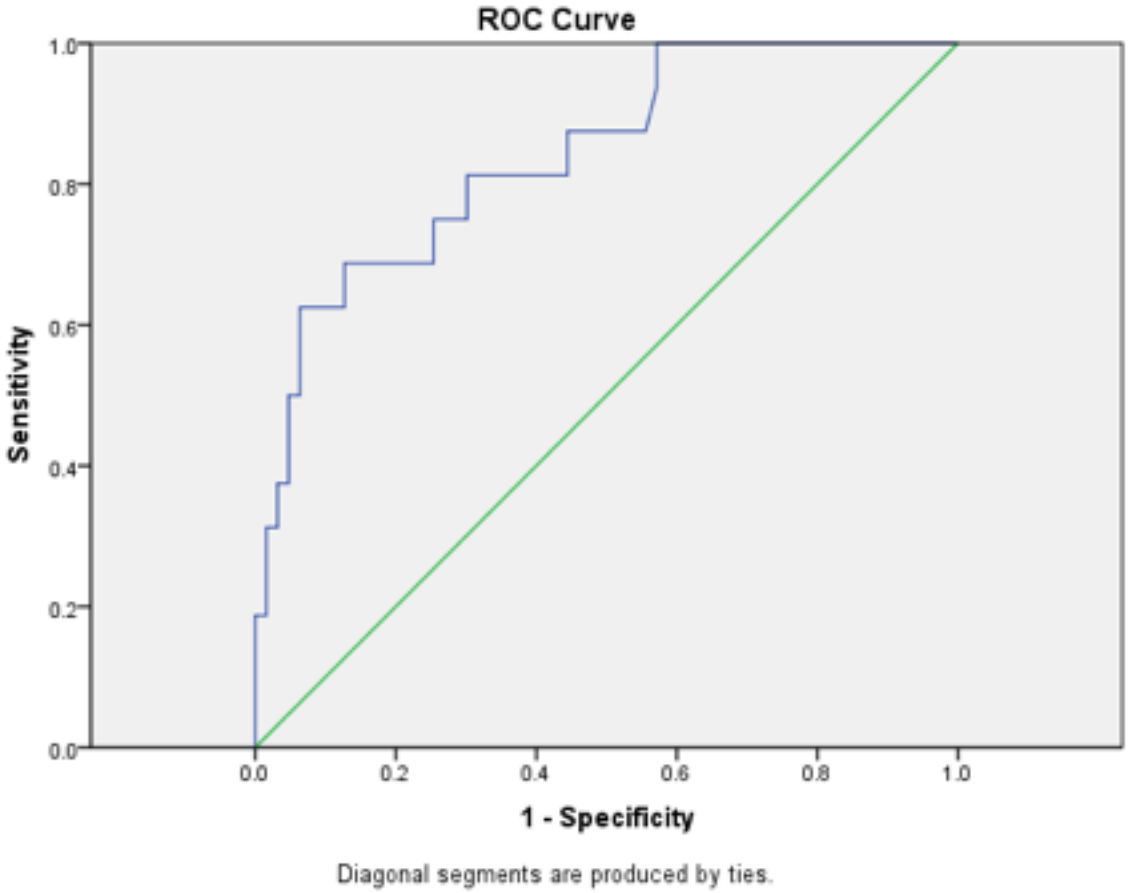


Area under curve – 0.878, p – 0.0001

Cut off – 24.75 cm, sensitivity – 87.5%, specificity – 73.0%

Cut off – 25.25 cm, sensitivity – 75.0%, specificity – 80.4%

Figure 10: ROC curve for Mid Arm Muscle Circumference (MAMC): Men

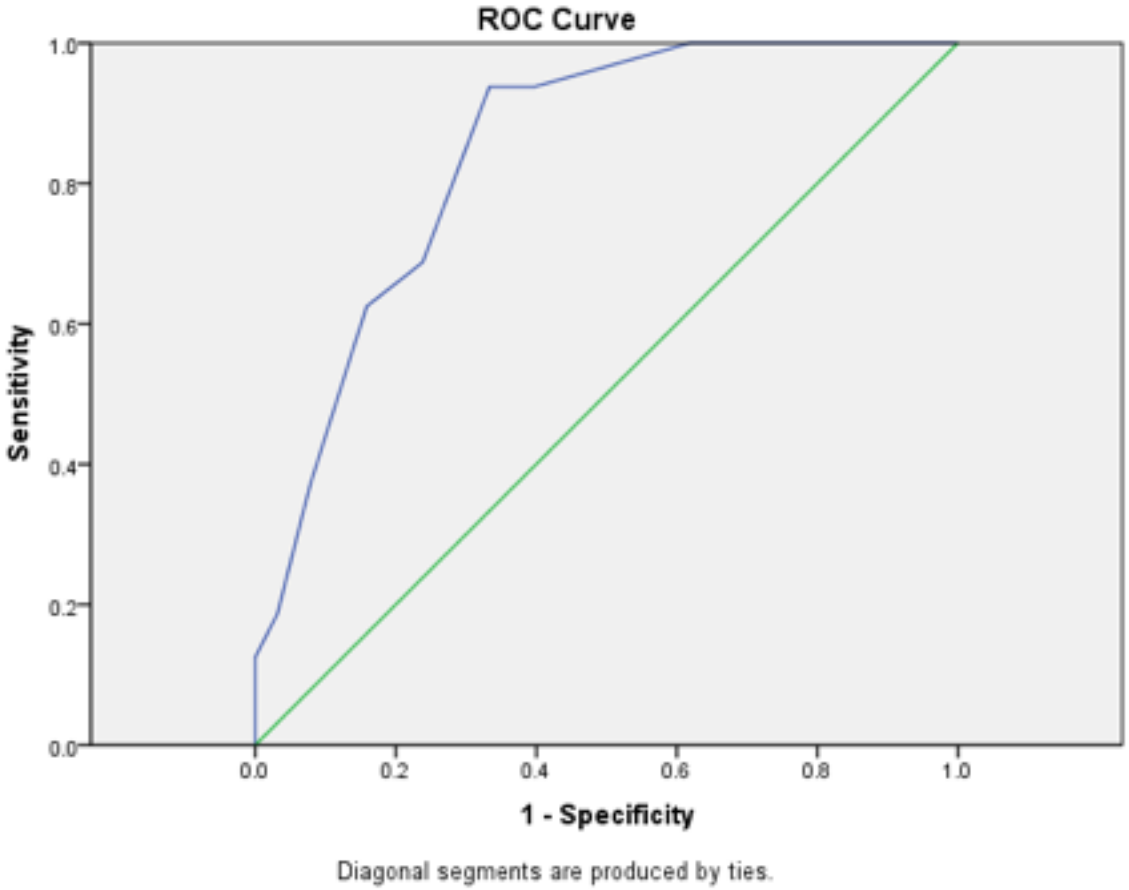


Area under curve – 0.841, p – 0.0001

Cut off – 21.93 cm, sensitivity – 81.3%, specificity – 69.8%

Cut off – 22.24 cm, sensitivity – 75.0%, specificity – 74.6%

Figure 11: ROC for Triceps Skin Fold Thickness (TSFT) : men

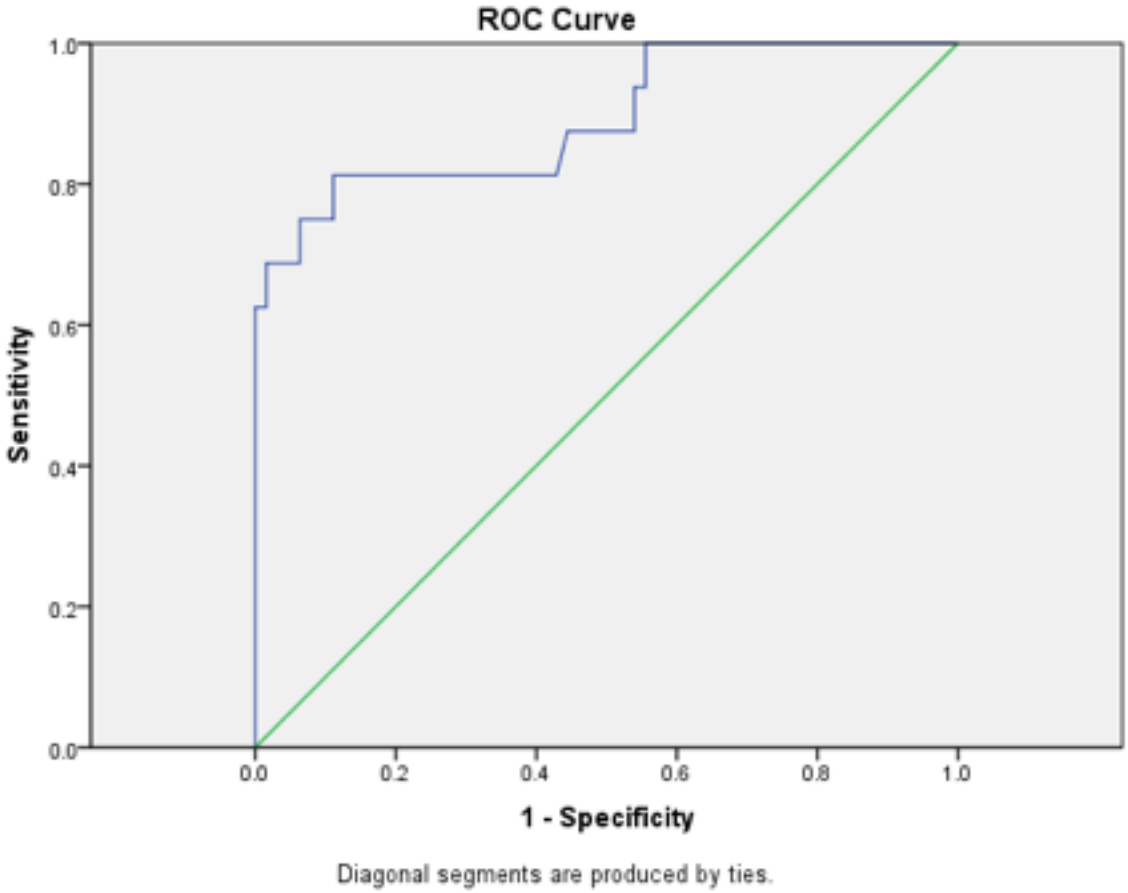


Area under curve – 0.843, p – 0.0001

Cut off – 9.50 mm, sensitivity – 93.8%, specificity – 66.7%

Cut off – 10.50 mm, sensitivity – 68.8%, specificity – 76.2%

Figure 12: ROC curve for Hand Grip Strength (male)



Area under curve – 0.892, p – 0.0001

Cut off – 27.90 Kg/F, sensitivity – 81.3%, specificity – 88.9%

Figure 13: Box Whisker plot for TSFT

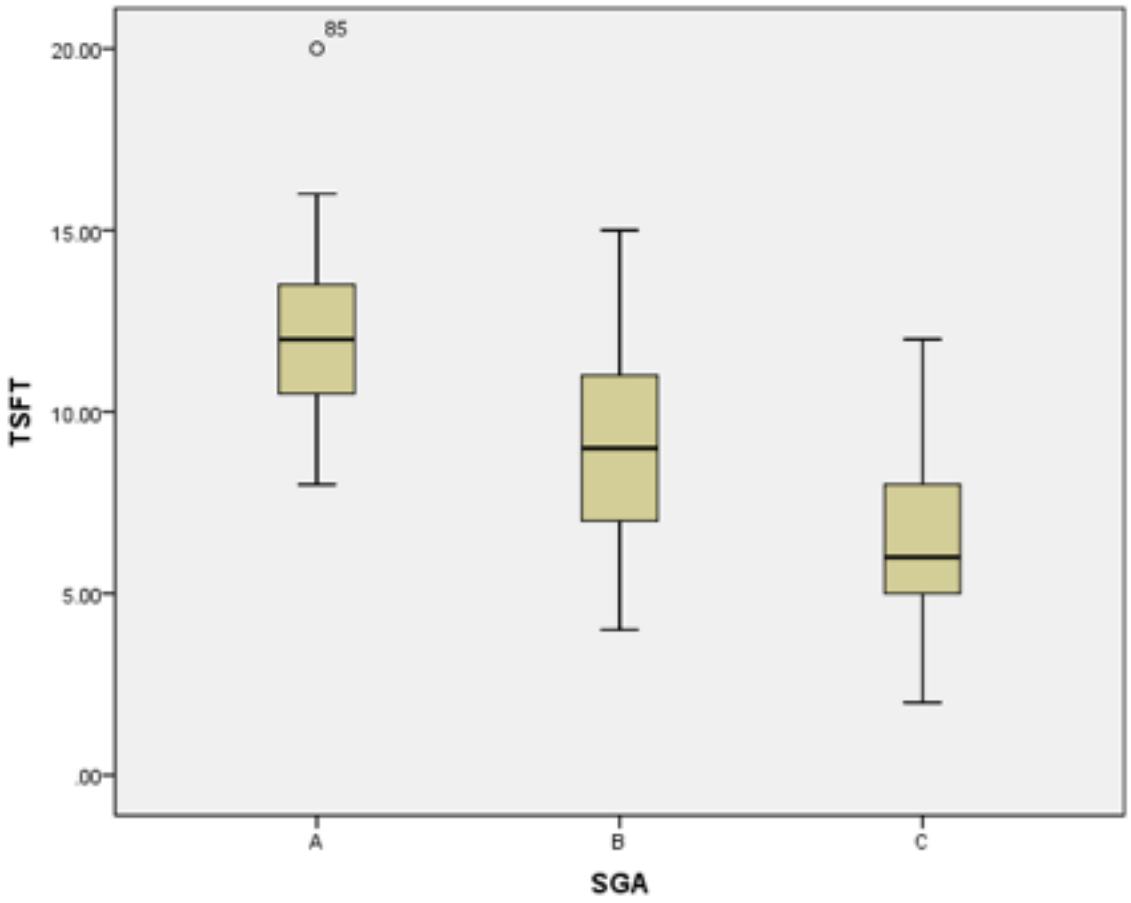


Figure 14: Box and Whisker plot for MAMC

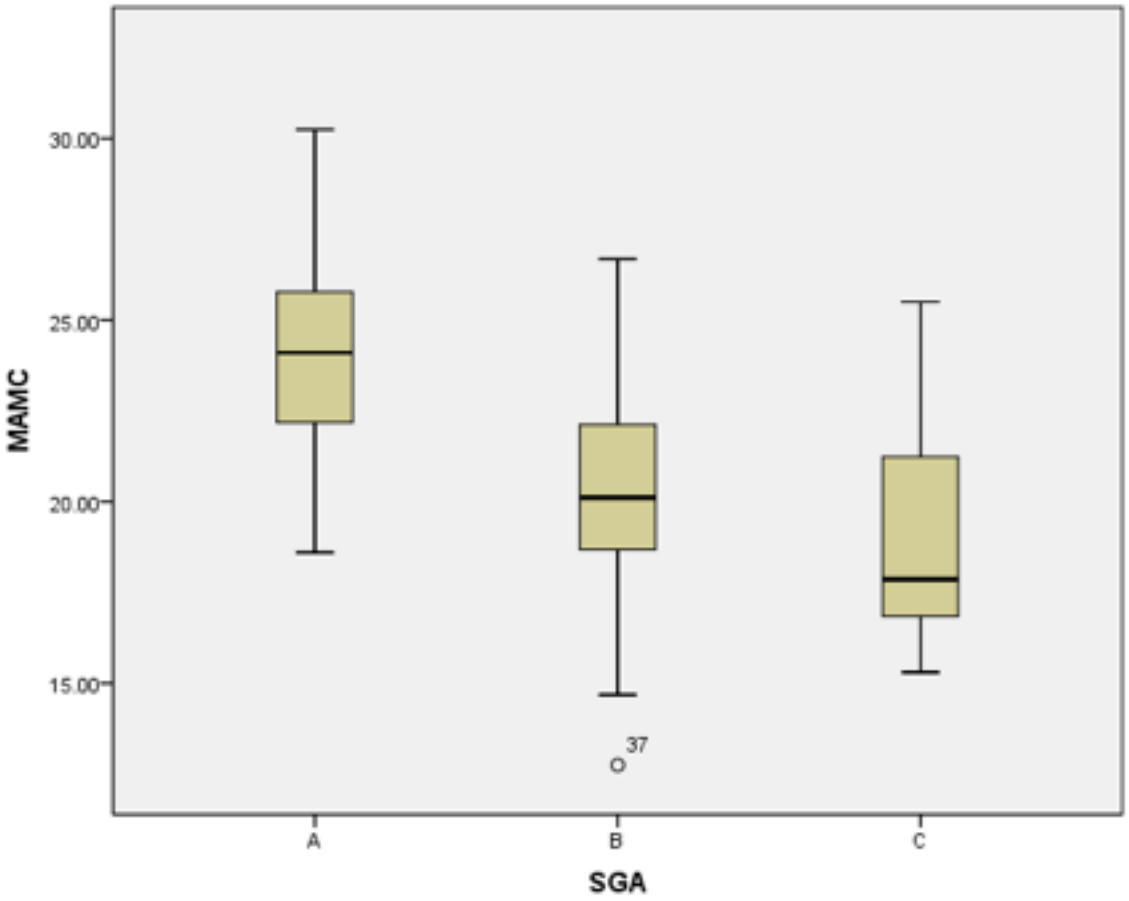
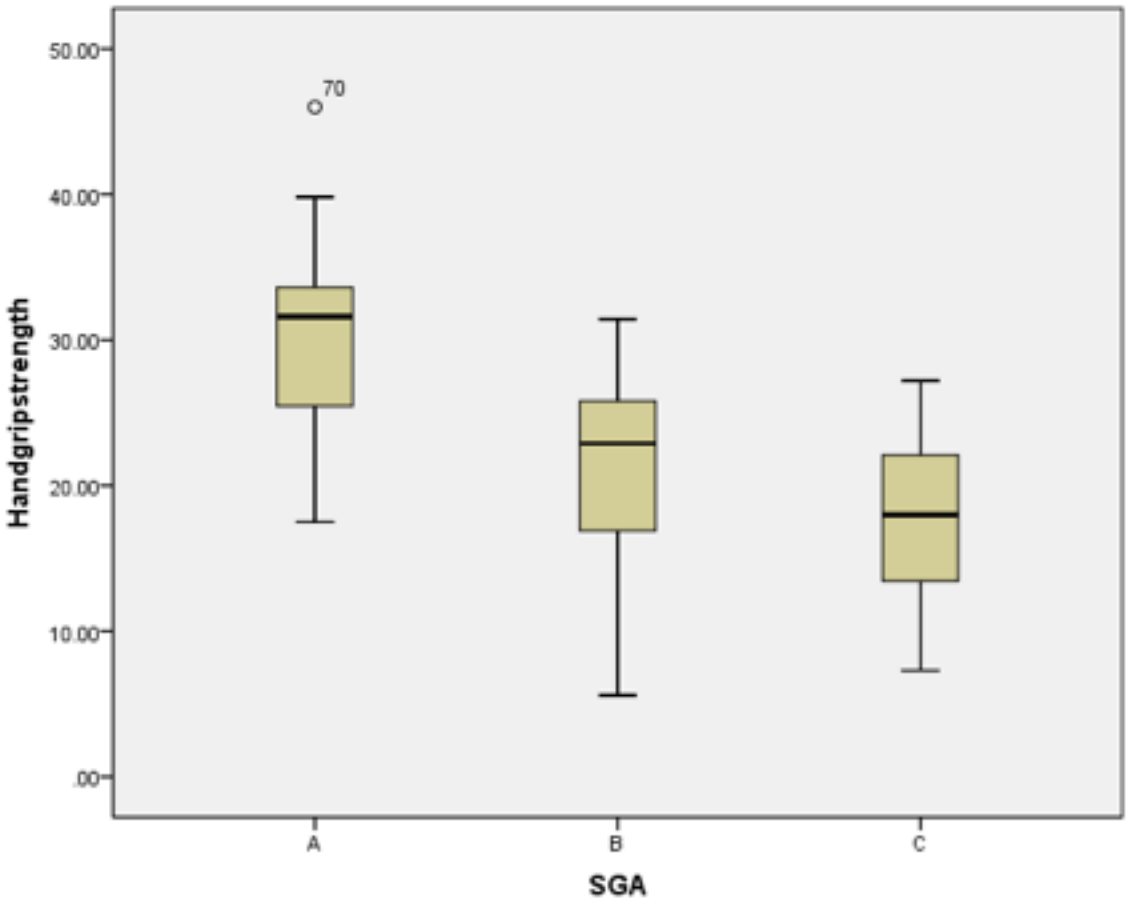


Figure 15: Box and Whisker plot for Hand Grip Strength



Biochemical and haematological parameters:

Hemoglobin level was not significantly different between different groups SGA A, SGA B and SGA C.

Table 11: ANOVA for Hemoglobin level between well nourished and malnourished

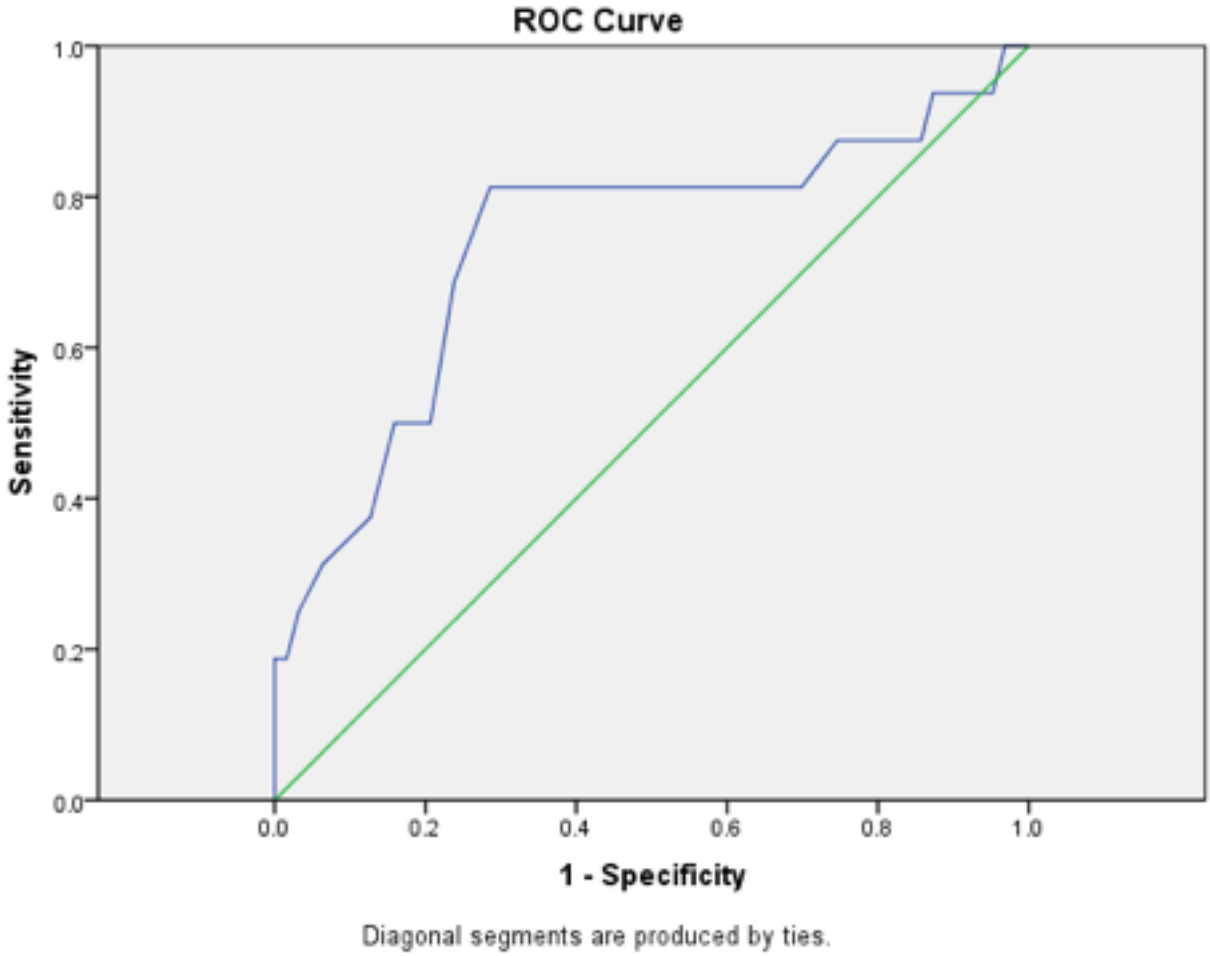
TEST	SGA A	SGA B	SGA C	p value
Hemoglobin g% (SE of mean)	9.74 (0.45)	9.13 (0.27)	8.54 (0.50)	(F-1.741) 0.182

The biochemical parameter Albumin was significantly low in SGA C and B compare to SGA A

Table 10:

Test	SGA A	SGA B	SGA C	p value
Mean Albumin in g % (SE of mean)	3.44 (0.17)	2.93 (0.09)	2.68 (0.15)	(F-6.36) 0.003

Figure 8: ROC for Albumin



Area under curve – 0.738, p – 0.003

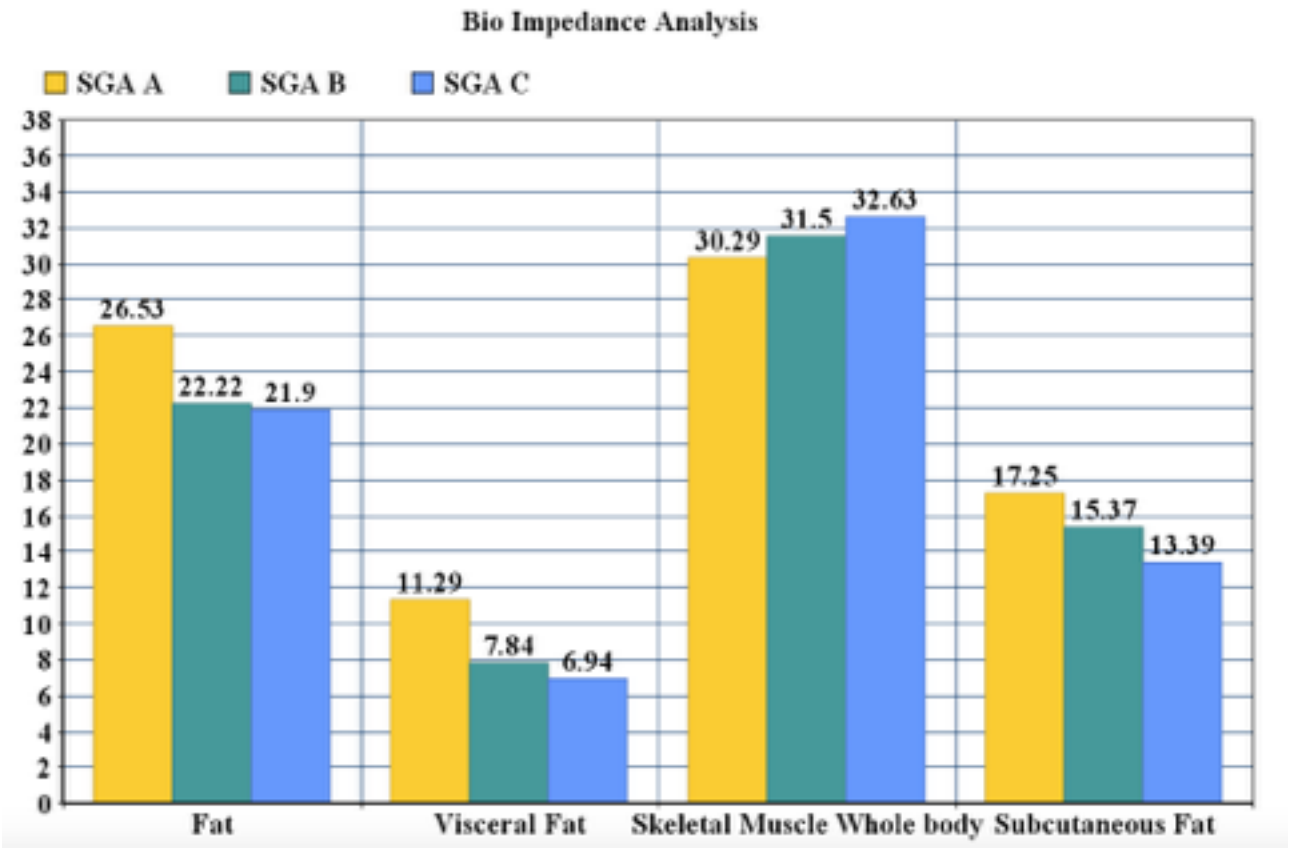
Cut off – 3.15 g%, sensitivity – 81.3%, specificity – 71.4%

Renal failure based on the lab data of Serum Creatinine value >1.4 mg/dl was seen in 6 patients (6/87, 6.89%). Among those with abnormal RFT 5 patients were malnourished, but this was not statistically significant to say that renal failure prevalence is more with malnourishment.

Bio Impedance Analyses:

The results obtained form bio impedance analyser were percentage of fat, visceral fat, skeletal muscle percentage (in whole body, trunk, arm, legs), subcutaneous fat percentage (whole body, trunk, arms, legs) and Resting Energy Expenditure (REE).

Figure 16: BIA values in different SGA group.



There was no significant difference in values of between SGA A, B and C in the values of whole body, visceral fat, skeletal muscle percentage, subcutaneous fat.

Table 12: ANOVA test for probability difference in BIA values between SGA A,B,C

Parameter	SGA A	SGA B	SGA C	P Value
Fat %	27.32 ± 2.39	22.95 ± 1.13	21.90 ± 2.91	(F-1.90) 0.159
Visceral fat %	11.15 ± 1.46	7.49 ± 0.81	6.95 ± 1.91	(F-2.77) 0.072

Resting Energy Expenditure (REE) was additional value obtained in the analyser. There was no significant difference between well nourished and malnourished patients taking REE.

Table 13: Resting Energy Expenditure in well nourished and malnourished

parameter	SGA A	SGA B	SGA C	p value
REE	1545.54 (65.08)	1456.18 (37.92)	1428.91 (68.39)	**(F-0.98) 0.382**

Complication in Cirrhosis:

The prevalence of Spontaneous Bacterial Peritonitis is 37.93%, Hepatic Encephalopathy is 25.2%. There was a significant increase in the prevalence of SBP (94.8% vs 5.2%, p= 0.0001, OR is 15.08) and Hepatic Encephalopathy (94.8% vs 5.2%, p= 0.0001, OR is 8.042) in Malnourished cirrhotics compared to well nourished patients. The prevalence of SBP (73.68% vs 34.69%, p=0.006) and HE (68.42% vs 16.32%, p=<0.0001) were higher in severely malnourished patients in comparison to patients with mild to moderate malnourishment. HE prevalence was high even though patients in encephalopathy of West Haven Criteria more than 1 were excluded form the study.

Table 14: Prevalence of complication compared to malnourishment

Complication	SGA A	SGA B	SGA C	p value
HE	1	8	13	(Chisquare-24.83) 0.0001
SBP	1	17	14	(Chisquare-19.34) 0.0001

Figure 17: Number of patients with complication of SBP and HE.

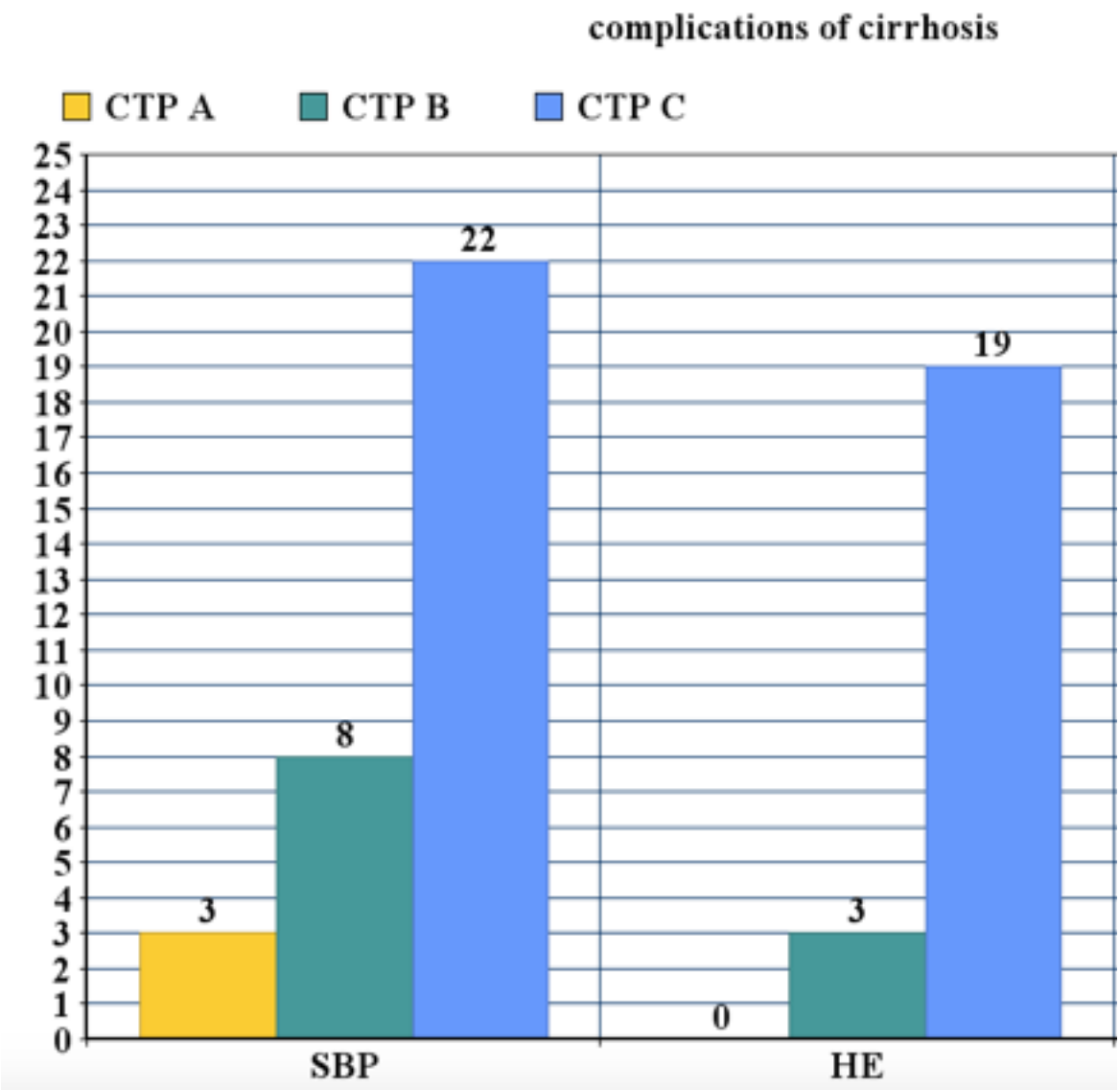
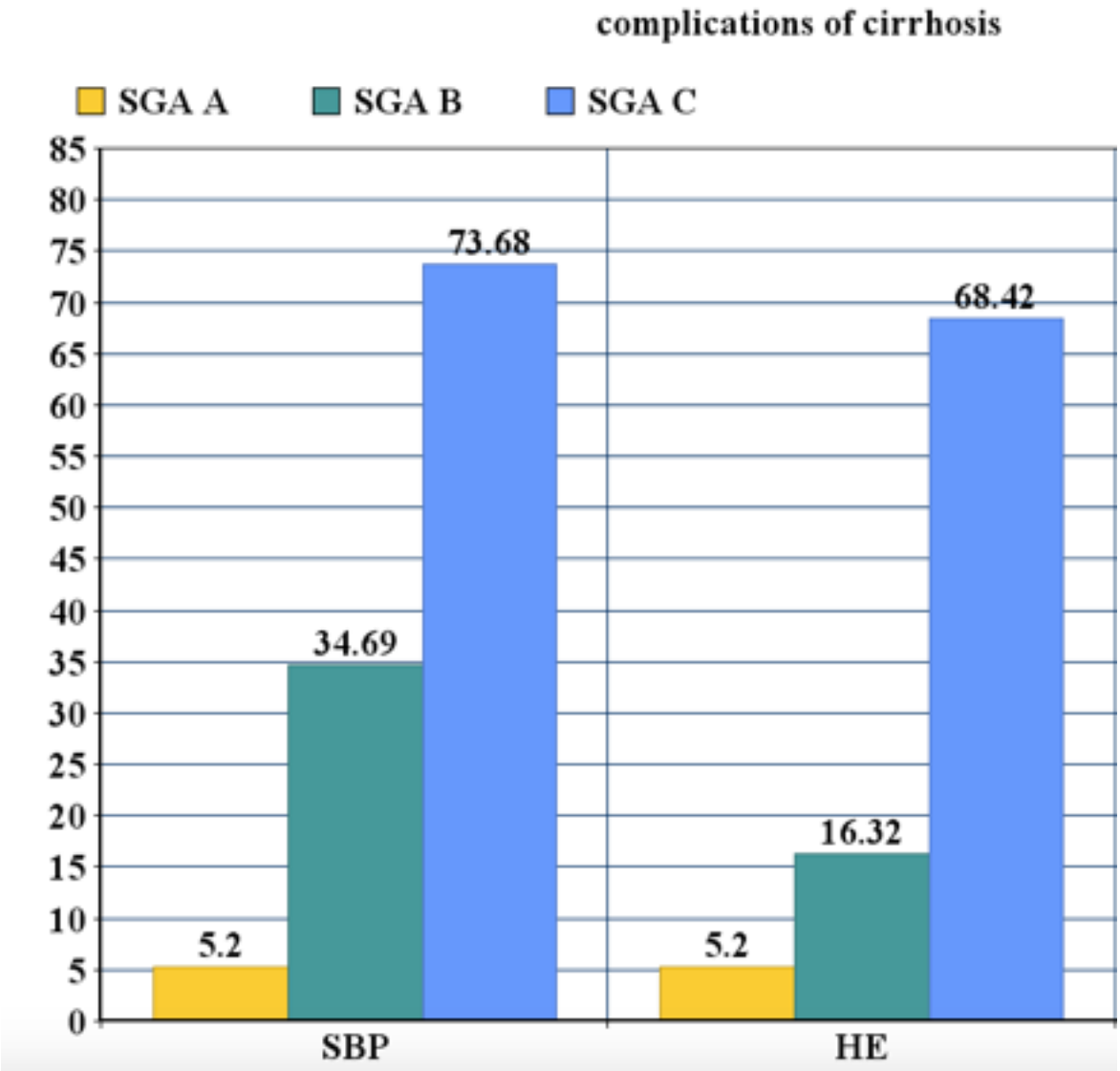


Figure 18: Prevalence of SBP and HE expressed in percentage.



Summary of Result:

	SGA			
Parameters	**A 19**	**B 49**	**C 19**	**p value**
BMI	25.29 (0.45)	22.47 (0.56)	22.27 (1.41)	**(F-3.092) 0.051**
Alcohol	9	36	18	***(Chisquare-10.73) 0.004***
HE	1	8	13	***(Chisquare-24.83) 0.0001***
SBP	1	17	14	***(Chisquare-19.34) 0.0001***
MAC (cm) mean ± 2 SE	27.66 (0.72)	23.06 (0.46)	21.13 (0.80)	***(F-21.06) 0.0001***
MAMC (mean ± 2 SE)	23.86 (0.69)	20.22 (0.39)	19.07 (0.70)	***(F-15.27) 0.0001***
TSFT (mm) (mean ± 2 SE)	12.32 (0.62)	9.06 (0.39)	6.57 (0.56)	***(F-21.98) 0.0001***
Handgrip (Kg/F) (mean ± 2 SE)	30.33 (1.66)	21.59 (0.86)	17.66 (1.26)	***(F-21.41) 0.0001***
Hemoglobin g% (mean ± 2 SE)	9.74 (0.45)	9.13 (0.27)	8.54 (0.50)	**(F-1.741) 0.182**
albumin g% (mean ± 2 SE)	3.44 (0.17)	2.93 (0.09)	2.68 (0.15)	***(F-6.36) 0.003***
fat % (Mean ± 2 SE)	27.32 (2.39)	22.95 (1.13)	21.90 (2.91)	**(F-1.90) 0.159**
V fat % (Mean ± 2 SE)	11.15 (1.46)	7.49 (0.81)	6.95 (1.91)	**(F-2.77) 0.072**
REE (Mean ± 2 SE)	1545.54 (65.08)	1456.18 (37.92)	1428.91 (68.39)	**(F-0.98) 0.382**

	SGA			
Parameters	**A**	**B**	**C**	**p value**
MAC male (cm) (Mean ± 2 SE)	28.00 (0.80)	23.39 (0.44)	21.13 (0.80)	***(F-22.05) 0.0001***
MAMC male (Mean ± 2 SE)	24.19 (0.74)	20.52 (0.38)	19.07 (0.70)	***(F-16.08) 0.0001***
TSFT male (mm) (Mean ± 2 SE)	12.38 (0.72)	9.14 (0.40)	6.58 (0.56)	***(F-20.64) 0.0001***
MAC female (cm) (Mean ± 2 SE)	25.83 (1.42)	20.20 (2.01)	-	(t-1.96)0.098
MAMC female (Mean ± 2 SE)	22.07 (1.74)	17.57 (1.50)	-	(t-1.894)0.107
TSFT female(mm) (Mean ± 2 SE)	12.00 (1.00)	8.40 (1.69)	-	(1.519)0.180

Discussion

In our study, only 87 patients were included as most of the patients were in overt encephalopathy and they were excluded. There was a clear gender bias, as 90% of the patients were male. The most common cause of cirrhosis in the study group was alcohol (72.8 %).

80.4% of the study group had decompensated cirrhosis with ascites.

The prevalence of malnutrition as per Subjective Global Assessment Score was 78.16%, which was in agreement with other studies. 21.8% of the study group were severely malnourished according to SGA score. Malnourishment is seen even in early cirrhosis of CTP class A.

Prevalence of Malnourishment in other studies[6,52,53]			
Author	**Centre, year**	**Study population**	**prevalence of malnourishment**
Patricia M Vieira	Brazil, 2013	78	61.5%
Mei-Ling S Tai	Malaysia, 2010	36	50%
Gunsar F	London, 2006	222	57%
Carvalho	Brazil, 2006	300	55%

Alcoholic patients were more prone for PEM according to literature. The fact that majority of the study group was alcoholics and majority of them had ascites, raised the prevalence of malnourishment in our study. Low socio economic background of all our patients could be an additional factor responsible for poor dietary intake. Similar increased PEM in alcoholics was reported by Carvalho et al[55]. Alcohol and Ascites both reduce the food intake. Salt restricted diet invariably was not well tolerated by our patients, and the possible effect of zinc deficiency secondary to diuretics could also be contributing to the decreased calorie intake. Similar to Carvalho et al study, our patients also showed significant malnourishment even in early stages of cirrhosis (CTP A). Hence Cirrhosis per se even if not decompensated is a cause for nutritional deficit through other mechanism of hyper metabolism, bile acid deficiency for micellar formation and fat absorption, muscle proteolysis for sustaining glycogen synthesis and others. Hence the nutritional parameters like Subjective Global Assessment score is invaluable in detecting the undernourished even early in the course of the disease, and it is probably the most cost effective bed side tool that is available so far.

The anthropometric values and albumin values significantly showed difference in the malnourished group (SGA B and C), whereas the BMI showed no significant difference and it was in the normal range even though the patients were malnourished. This highlights the intention of the this study to identify additional simple bedside parameters to diagnose malnourishment in cirrhosis, as BMI is not the reliable test when the patient is volume

overloaded. Ascites and oedema adds to the weight of the patient. Using SGA score, mild to moderate malnourishment was detected in 29.4% of the patients who had no ascites.

The normal range for anthropometry values has not been defined in the healthy population in coastal Tamilnadu before. We took the measurements from healthy volunteers and found that the normal mean values for MAMC, TSFT, and Hand grip strength were 25.24 cm, 15.6 cm, and 38.7 Kg/F respectively in males, and 23.36 cm, 21 cm and 25.56 Kg/F respectively in females.

MAMC and Handgrip strength reference values were in agreement with reference values of Malaysia StudyTai et al[52] , but TSFT reference values were 5 mm for males and 12 mm fro females which was less than our reference population in coastal Tamilnadu.

The anthropometry values were significantly low in malnourished group only in male patients and not in females as the sample size was too low to extrapolate the findings to end result. The MAMC was 24.19 + 0.74 cm, 20.52 $\pm$ 0.38 cm and 19.07 $\pm$ 0.70 cm in SGA A, B and C respectively (p= 0.0001, F-16.08). The cut off identified to diagnose malnourishment in cirrhosis in male in our study was 22.24 cm (sensitivity – 75.0%, specificity – 74.6%, AUROC 0.841).

Triceps Skin Fold Thickness was 12.38 $\pm$ 0.72, 9.14 $\pm$ 0.40, and 6.58 $\pm$ 0.56 in SGA A, B and C respectively (p=0.0001, F-20.64). The cut off value of

TSFT for diagnosis malnourishment is 9.50 mm (sensitivity – 93.8%, specificity – 66.7%, AUROC 0.843).

Hand Grip Strength was 30.33 ± 1.66 Kg/F, 21.59 ± 0.86 Kg/F and 17.66 ± 1.26 Kg/F in SGA A, B and C respectively (p= 0.0001, F-21.41). The cut value of Hand Grip Strength for diagnosing malnourishment in our study was 27.90 Kg/F (sensitivity – 81.3%, specificity – 88.9%, AUROC 0.892).

Patricia et al[53] in Brazil and Gulzar et al[6] in London, produced similar MAMC and TSFT values in malnourished patients. Those studies did not use Hand Grip Dynamometry.

Tai et al[52] in Malaysia used Hand Grip Dynamometry in population of 36 patients and did not find any significant difference in Hand Grip values between well nourished and malnourished patients.

Hence the cost effective bed side assessment parameters like Mid Arm Muscle circumference, Triceps Skin fold Thickness and Hand Grip strength can detect undernourished group of cirrhotics early in the course of disease to subject them to medical nutritional therapy.

The Bio Impedance Analyses is a valuable tool in assessing nutrition. The principle involved is passing minute electric current through the body via electrodes, and reading the resistance to the flow with another electrode. Fat and bone offers more impedance to current flow, whereas electrolyte dissolved water conducts the current faster. Relative proportion of body compartment can be measured. Ideal parameter measured to diagnose

malnourishment according to Fernandes et al[54] was phase angle. Phase angle is the proportion of resistance to capacitance which reflects the contribution of fluid and cell membrane of the body. A value less than 5.4^{o} was found to significant in detecting malnourishment. The bio impedance values in our study did not show significant change in body composition with malnourishment. This finding reiterates that the principle of BIA faces debacle when used in volume redistributed conditions like cirrhosis.

The prevalence of SBP and Hepatic Encephalopathy was significantly high in malnourished patients. In malnourished cirrhotics, the Odds ratio for developing SBP was 15.08 and Odds ratio for HE was 8.042. This was in agreement with experimental study from Casafont et al[26], which states that the risk of developing SBP is more as Bacterial translocation into ascites is more with malnourished rats. Decompensated cirrhosis patients subjected to repeated paracenteses and diuresis lose the macronutrients from their ascitic fluid. They invariably progress towards the course of nutrient deficit. Hence it is inevitable that infections like SBP is going to be a major problem in malnourished cirrhotics. Unless long term antibiotic prophylaxis for SBP and medical nutrition therapy becomes a part of standard of care, the therapeutic management may be considered as incomplete in malnourished and decompensated cirrhotic patients .

Hepatic Encephalopathy is historically associated with deranged protein metabolism. The prevalence of Hepatic Encephalopathy being high with malnourishment in our study was in agreement with the study by Kalaitzakis et al[56], where he had concluded poor nutritional status as the pathogenetic mechanism for HE.

Low protein diet was not warranted in the past for HE, but as the knowledge regarding the nutritional status in cirrhosis has evolved to account protein calorie deficit as the major nutrient deficit, withholding protein is not the standard of care anymore. Excess of aromatic amino acids and deficit in essential amino acids like BCAA (lysine, leucine and isoleucine) is seen in cirrhosis.

Protein supplementation is graded in cirrhosis with balance tipped towards Branched amino acids, and protein should not be restricted to <1g/kg BW.

So all our patients were given a proper diet advise which consisted of a high calorie diet of 30-40 K Cal/day inclusive of 1.5 g/kg protein/day, with special mention about evening snack, maintenance of inter prandial period to less than 4 hours.

Conclusion:

1) The prevalence of malnourishment in cirrhosis is 78.16 %, of which severe malnourishment is seen in 21.8%

2) Subjective Global Assessment Score is very useful in diagnosing malnourishment

3) Anthropometric parameters like Triceps skin fold thickness, Mid Arm Circumference, and Mid Arm Muscle Circumference are useful bedside tests in diagnosing malnourishment

4) Hand Grip Dynamometry is very useful in diagnosing malnourishment and categorising the severity of malnourishment in cirrhosis.

5) Bio Impedance Analyser is not useful in categorising the nourishment status of decompensated cirrhosis patients.

6) Malnutrition is a risk factor for developing Spontaneous Bacterial Peritonitis and Hepatic Encephalopathy.

Limitation:

The study population has selection bias as female patients were too low to draw conclusion from the results. Further study on a larger population is needed to validate the finding in this study.

The Bio Impedance Analyser used in our study has not been used as a standard in any previous study before.

Bibliography

1) Lozano R, Naghavi M, Foreman K, Lim S, Shibuya K, Aboyans V, Abraham J, Adair T, Aggarwal R, Ahn SY, Alvarado M, Anderson HR, Anderson LM, Andrews KG, Atkinson C, Baddour LM, Barker-Collo S, Bartels DH, Bell ML, Benjamin EJ, Bennett D, Bhalla K, Bikbov B, Bin Abdulhak A, Birbeck G, Blyth F, Bolliger I, Boufous S, Bucello C, Burch M, et al.: Global and regional mortality from 235 causes of death for 20 age groups in 1990 and 2010: a systematic analysis for the Global Burden of Disease Study 2010, Lancet 2012.

2) Kondrup J,Muller M J. Energy and protein requirement of patients with chronic liver disease. J Hepatol 1997;27:239 – 47.

3) Peng S, Plank LD, McCall JL, Gillanders LK, McIlroy K, Gane EJ. Body composition, muscle function, and energy expenditure in patients with liver cirrhosis: a comprehensive study. *Am J Clin Nutr*. 2007;85(5):1257-1266.

4) Krall EA, Dwyer JT. Validity of a food frequency questionnaire and a food diary in a short-term recall situation. *J Am Diet Assoc*. 1987;87(10): 1374-1377

5) Nutrition Assessment and Management in Advanced Liver Disease, Tammy M. Johnson et al; *Nutr Clin Pract*. 2013;28:15-29

6) Nutritional status and prognosis in cirrhotic patients ,F. Gunsar, M. L. Raimondo, S. Jones , N. Terreni, C. Wong, D. Patch, C. Sabinà & A. K. Burroughs, Aliment Pharmacol Ther 24, 563–572

7) Nolte W, Hartmann H, Ramadori G: Glucose metabolism and liver cirrhosis. Exp Clin Endoscrinol Diabetes. 103:63-74 1995

8) Cicogni C, Malavolti M, Morselli-Labate AM, et al.: Serum lipid and lipoprotein patterns in patients with cirrhosis and chronic active hepatitis. Arch Intern Med. 157:792-796 1997

9) Look MP, Reichel C, von Falkenhausen M, et al. Vitamin E sta- tus in patients with liver cirrhosis: normal or deficient? *Metabolism*. 1999;48(1): 86-91. 69.

10) Baker H, Leevy CB, DeAngelis B, Frank O, Baker ER. Cobalamin (vitamin B12) and holotranscobalamin changes in plasma and liver tissue in alcoholics with liver disease. J Am Coll Nutr. 1998;17(3):235-238

11) Halifeoglu I, Gur B, Aydin S, Ozturk A. Plasma trace elements, vitamin B12, folate, and homocysteine levels in cirrhotic patients compared to healthy controls. *Biochemistry (Mosc)*. 2004;69(6):693-696.

12)PengS,PlankLD,McCallJL,GillandersLK,McIlroyK,GaneEJ.Body composition, muscle function, and energy expenditure in patients with liver cirrhosis: a comprehensive study. Am J Clin Nutr 2007;85:1257– 66

13)Schatzkin A, Kipnis V, Carroll RJ, et al. A comparison of a food frequency questionnaire with a 24-hour recall for use in an epidemiologi- cal cohort study: results from the biomarker-based Observing Protein and Energy Nutrition (OPEN) study. *Int J Epidemiol*. 2003;32(6):1054- 1062.

14)Chang WT, Ker CG, Hung HC, et al. Albumin and prealbumin may predict retinol status in patients with liver cirrhosis. *Hepatogastroenterology*. 2008;55(86-87):1681-1685.

15)Fiore P, Merli M, Andreoli A, et al. A comparison of skinfold anthropometry and dual-energy X-ray absorptiometry for the evaluation of body fat in cirrhotic patients. *Clin Nutr*. 1999;18(6):349-351.

16)Fernandes SA, Bassani L, Nunes FF, Aydos ME, Alves AV, Marroni CA. Nutritional assessment in patients with cirrhosis. *Arq Gastroenterol*. 2012;49(1):19-27.

17)Fields D, Goran M, McCrory M. Body composition assessment via air-displacement plethysmography in adults and children: A review. Am J Clin Nutr 2002; 75:453-67.

18)Genton L, Hans D, Kyle U, et al. DEXA and body composition: Differences between devices and comparison with reference methods. Nutrition 2002; 18:66-70.

19) Kehayias J, Fiatarone M, Zhuang H, et al. Total body potassium and fat: Relevance to aging. Am J Clin Nutr 1997; 66:904-10.

20) Schoeller D Kushner R, Taylor P, et al. Measurement of total body water: Isotope dilution techniques. In: Roche A, editor. Body composition assessments in youth and adults. 6th ed. Columbus, Ohio: Ross Laboratories; 1985. p 24.

21) Ellis K, Bell S, Chertow G, et al. Bioelectrical impedance methods in clinical research: A follow-up to the NIH technology assessment conference. Nutrition 1999; 15:874-80.

22)Figueiredo F, Dickson E, Pasha T, et al. Utility of standard nutritional parameters in detecting body cell mass depletion with end-stage liver disease. Liver Transpl 2000; 6:575-81.

23)Nascimento M, Qureshi A, Stenvinkel P, et al. Malnutrition and inflammation are associated with impaired pulmonary function in patients with chronic kidney disease. Nephrol Dial Transplant 2004; 19:1823-8.

24)Garcia-Tsao G, Friedman S, Iredale J, et al. Now there are many (stages) where before there was one: In search of a pathophysiological classification of cirrhosis. Hepatology 2010; 51:1445-9.

25)Mason JB. Gastrointestinal cancer; nutritional support. In: Kelsen D, Daly J, Kern S, et al, editors. Principles and practice of gastrointestinal oncology. Philadelphia: Lippincott Williams & Wilkins; 2002

26)Casafont et al. Influence of malnutrition on the prevalence of bacterial translocation and spontaneous bacterial peritonitis in experimental cirrhosis in rats. Hepatology. 1997 Jun;25(6):1334-7.

27)B. Campillo, J. P. Richardet, and P. N. Bories, “Enteral nutrition in severely malnourished and anorectic cirrhotic patients in clinical practice: benefit and prognostic factors,” Gastroenterologie Clinique et Biologique, vol. 29, no. 6-7, pp. 645–651, 2005

28)T. L. Sourkes, “Tryptophan in hepatic coma,” Journal of Neural Transmission, Supplement, no. 14, pp. 79–86, 1978

29) Sleisenger and Fordtran’s Gastro Intestinal and Liver Diseases 10th edition, Chapter 5 page 75.

30)Cordoba J, Lopez-Hellin J, Planas M, et al. Normal protein diet for epi-sodic hepatic encephalopathy: results of a randomized study. *J Hepatol*. 2004;41(1):38-43

31)Mueller CM. The American Society for Parenteral and Enteral Nutrition (A.S.P.E.N.) Adult Nutrition Support Core Curriculum. 2nd ed. Sil- ver Spring, MD: American Society for Parenteral and Enteral Nutrition; 2012.

32)G. P. Bianchi, G. Marchesini, A. Fabbri et al., “Vegetable versus animal protein diet in cirrhotic patients with chronic encephalopathy. A randomized cross-over comparison,” Journal of Internal Medicine, vol. 233, no. 5, pp. 385–392, 1993

33)Plauth M, Cabre E, Campillo B, et al. ESPEN Guidelines on Parenteral Nutrition: hepatology. *Clin Nutr*. 2009;28(4):436-444.

34)Gabe SM, Culkin A. Abnormal liver function tests in the parenteral nutrition fed patient. *Frontline Gastroenterology*. 2010(1):98-104.

35)M. Iwasa, K. Matsumura, Y. Watanabe et al., “Improvement of regional cerebral blood flow after treatment with branched-chain amino acid solutions in patients with cirrhosis,” European Journal of Gastroenterology and Hepatology, vol. 15, no. 7, pp. 733–737, 2003.

36)Sorrentino P, Castaldo G, Tarantino L, et al. Preservation of nutritional-status in patients with refractory ascites due to hepatic cirrho- sis who are undergoing repeated paracentesis. *J Gastroenterol Hepatol*. 2012;27(4): 813-822.

37)G. Marchesini, G. Bianchi, M. Merli et al., “Nutritional supplementation with branched-chain amino acids in advanced cirrhosis: a double-blind, randomized trial,” Gastroenterology, vol. 124, no. 7, pp. 1792–1801, 2003.

38)Y. Nakaya, K. Okita, K. Suzuki et al., “BCAA-enriched snack improves nutritional state of cirrhosis,” Nutrition, vol. 23, no. 2, pp. 113–120, 2007

39)Y. Muto, S. Sato, A. Watanabe et al., “Effects of oral branched-chain amino acid granules on event-free survival in patients with liver cirrhosis,” Clinical Gastroenterology and Hepatology, vol. 3, no. 7, pp. 705–713, 2005

40)M. Kugelmas, D. B. Hill, B. Vivian, L. Marsano, and C. J. McClain, “Cytokines and NASH: a pilot study of the effects of lifestyle modification and vitamin E,” Hepatology, vol. 38, no. 2, pp. 413–419, 2003

41)M. P. de la Maza, M. Petermann, D. Bunout, and S. Hirsch, “Effects of long-term vitamin E supplementation in alcoholic cirrhotics,” Journal of the American College of Nutrition, vol. 14, no. 2, pp. 192–196, 1995

42) C. Bémeur, J. Vaquero, P. Desjardins, and R. F. Butterworth, “N-acetylcysteine attenuates cerebral complications of non-acetaminophen-induced acute liver failure in mice: antioxidant and anti-inflammatory mechanisms,” Metabolic Brain Disease, vol. 25, no. 2, pp. 241–249, 2010

43) S. Holt, D. Goodier, R. Marley et al., “Improvement in renal function in hepatorenal syndrome with N-acetylcysteine,” Lancet, vol. 353, no. 9149, pp. 294–295, 1999

44) Arthur J Patek Jr, Treatment Of Cirrhosis Of The Liver By A Nutritious Diet And Supplements Rich In Vitamin B Complex;J Clin Invest. 1941 Sep; 20(5): 481–505.

45) Satish Et Al, Phase Angle Measurement In Healthy Human Subjects Through Bio-Impedance Analysis: Iran J Basic Med Sci. 2012 Nov-Dec; 15(6): 1180–1184

46) Q. Liu, Z. P. Duan, D. K. Ha, S. Bengmark, J. Kurtovic, And S. M. Riordan, “Synbiotic Modulation Of Gut Flora: Effect On Minimal Hepatic Encephalopathy In Patients With Cirrhosis,” Hepatology, Vol. 39, No. 5, Pp. 1441–1449, 2004.

47) W A S Peres, Phase Angle As A Nutritional Evaluation Tool In All Stages Of Chronic Liver Disease. Nutr. Hosp. Vol.27 No.6 Madrid Nov.-Dec. 2012

48) Choban PS, Flancbaum L. Nourishing the obese patient. *Clin Nutr*. 2000;19(5):305-311.

49)Petrides AS, Vogt C, Schulze-Berge D, Matthews D, Strohmeyer G. Pathogenesis of glucose intolerance and diabetes mellitus in cirrhosis. *Hepatology.* 1994;19(3):616-627

50)Suzuki A, Lindor K, St Saver J, et al. Effect of changes on body weight and lifestyle in nonalcoholic fatty liver disease. *J Hepatol*. 2005;43(6): 1060- 1066.

51) Hasse J, Strong S, Gorman MA, Liepa G. Subjective global assessment: alternative nutrition-assessment technique for liver-transplant candidates. Nutrition 1993; 9: 339-343.

52)Tai et al.:Anthropometric, biochemical and clinical assessment of malnutrition in Malaysian patients with advanced cirrhosis, Nutrition Journal 2010, **9**:27

53) Patrici M Vieira,Nutritional assessment in hepatic cirrhosis; clinical, anthropometric, biochemical and hematological parameters. Nutr Hosp. 2013;28:1615-1621

54) Fernandes SA, Bassani L, Nunes FF, Aydos MED, Alves AV, Marroni CA. Nutritional assessment in patients with cirrhosis. Arq gastroenterol. 2012; 49:19-27.

55) Carvalho l, Parise ER. Evaluation of nutritional status of non hospitalized patients with liver cirrhosis. Arq gastroenterol. 2006; 43: 269-74.

56) Kalaitzakis E, Malnutrition and diabetes mellitus are related to hepatic encephalopathy in patients with liver cirrhosis. Liver Int. 2007 Nov;27(9): 1194-201.

57) Garcia-Tsao G, Friedman S, Iredale J, et al. Now there are many (stages) where before there was one: In search of a pathophysiological classification of cirrhosis. Hepatology 2010; 51:1445-9.

58) Mifflin M, St. Jeor S, Hill L, et al. A new predictive equation for resting energy expenditure in healthy individuals. Am J Clin Nutr 1990; 51:241-7.

ANNEXURE

PROFORMA

Name Age Sex
DDHD no

Diagnosis Aetiology

CTP score MELD

USG

Hb Platelets Urea Creatinine P T control INR Bilirubin Albumin
Ascites Neurological status

Anthopometry

Weight Height BMI M i d A r m circumference Triceps skin fold thickness (TSFT)

Mid Arm Muscle Circumference (MAMC)

Hand Grip Strength (average of 3 reading)

BIE:

Body fat Visceral fat REE

Sub Cutaneous Fat : Whole Body Trunk Arm Legs

Skeletal Muscle : Whole Body Trunk Arm Legs

Subjetive Global Assessment:

SBP HE GI bleed

Subjective Global Assessment

Name:
Date:

Medical History			A	B	C
WEIGHT	Usual weight.....	Current weight.....			
Wt change past 6 months	Amount weight loss.....	% weight loss.....			
0-<5% loss			*		
5-10% loss				*	
>10% loss					*
Weight change past 2 weeks		Amount.....			
No change; normal weight			*		
Increase to within 5%			*		
Increase (1 level above)			*	*	
No change, but below usual wt				*	
Increase to within 5-10%				*	
Decrease					*
DIETARY INTAKE					
No change; adequate			*		
No change; inadequate				*	
Change	**Duration of change**.....				
Suboptimal diet				*	
Full liquid				*	
Hypocaloric liquid					*
Starvation					*
Intake borderline; increasing			*		
Intake borderline; decreasing				*	
Intake poor; no change				*	*
Intake poor; increasing				*	
Intake poor; decreasing					*
GASTROINTESTINAL SYMPTOMS					
	Frequency (never, daily, no. of times/week)	Duration (<2wk, >2wk)			
Nausea	.....	.....			
Vomiting	.....	.....			
Diarrhoea	.....	.....			
Anorexia	.....	.....			
None; intermittent			*		
Some (daily >2 week)				*	
All (daily >2 week)					*
FUNCTIONAL CAPACITY					
No dysfunction	Duration of change		*		
Difficulty with ambulation/normal activities				*	
Bed/chair-ridden					*
Change past 2 week					
Improved			*		
No change				*	
Regressed					*

Physical examination	A	B	C
SUBCUTANEOUS FAT			
Under the eyes	Slightly bulging area		Hollowed look, depression, dark circles
Triceps	Large space between fingers		Very little space between fingers, or fingers touch
Biceps	Large space between fingers		Very little space between fingers, or fingers touch
MUSCLE WASTING			
Temple	Well-defined muscle/flat	Slight depression	Hollowing, depression
Clavicle	Not visible in Males; may be visible but not prominent in females	Some protrusion; may not be all the way along	Protruding/prominent bone
Shoulder	Rounded	No square look; acromion process may protrude slightly	Square look; bones prominent
Scapula/ribs	Bones not prominent; no significant depressions	Mild depressions or bone may show slightly; not all areas	Bones prominent; significant depressions
Quadriceps	Well rounded; no depressions	Mild depression	Depression; thin
Calf	Well developed		Thin; no muscle definition
Knee	Bones not prominent		Bones prominent
Interosseous muscle between thumb and forefinger	Muscle protrudes; could be flat in females		Flat or depressed area
OEDEMA (related to malnutrition)	No sign	Mild to moderate	Severe
ASCITES (related to malnutrition)	No sign	Mild to moderate	Severe
OVERALL SGA RATING	**A**	**B**	**C**

Adapted from: Detsky et al., 1994[8]; Baxter Healthcare Corporation, 1993; McCann, 1996 (Ferguson, Bauer, Banks, Capra, 1996)©

nam	ddhd	age	sex	etiol	dura	CTP	HE	Blee	SBP	biliru	albu	INR	ascit	urea	crea	Hb	heigl	weig	BMI	MAC	TSFT	MAM	Hanc	SGA
pasu	1794	5	1	1	1	2	1	1	1	2.3	3.1	1.7	3	27	0.7	9	160	62	24.2	22	10	18.8	12.6	2
govir	1020	3	1	1	2	2	1	2	1	2.6	2.5	1.3	3	16	0.6	9	169	72	25.2	27.5	8	24.9	36	1
shan	957.	3	1	1	1	3	1	1	2	6.5	2	2.3	3	18	0.6	8.6	166	45	16.3	17.5	4	16.2	10.3	3
raju	7426	4	1	1	2	2	1	1	2	1.1	3.4	1.5	3	28	0.8	11.8	163	57.3	21.6	25.5	11	22.0	24	2
veer	3380	5	1	1	2	3	1	1	2	7.5	0.7	2.4	4	19	0.6	8.5	163	56	21.1	16	2	15.3	7.3	3
mani	875.	6	1	1	3	2	1	1	1	2.5	3.5	1.3	3	36	1.5	11.4	169	75	26.3	19	6	17.2	19.3	3
chinr	919.	5	1	1	3	3	1	1	2	4.5	2.8	2.3	4	19	0.7	8.6	155	63	26.2	24	4	22.7	12.6	3
sridh	5802	4	1	1	2	2	1	1	2	1.3	3.7	1.3	4	42	1.1	10.2	147	45	20.5	20.5	8	18	5.6	2
nivec	2473	2	2	4	3	1	1	2	1	1.9	4.1	1.2	1	19	0.7	7.9	170	70	24.2	27.5	11	24.0	28	1
arpu	1089	4	1	1	2	2	1	1	1	1.1	3.3	1.2	3	40	1.1	11.8	165	63	23.1	29	12	25.4	32.6	1
chan	3279	6	1	1	2	2	1	1	2	1.1	3.1	1.1	3	16	0.8	12	163	48	18.1	19	8	16.4	14	2
sasik	7635	4	1	1	2	3	2	1	2	5	1.9	2.1	4	22	0.6	6.5	156	95	39	28	8	25.5	13	3
krish	6868	5	2	2	3	2	1	1	2	1.1	3.2	1.6	1	25	0.7	11.2	150	56	24.8	24	12	20.2	16.3	2
sriniv	484.	5	1	1	1	2	2	1	1	3.3	2.1	1.6	3	14	0.5	11.6	167	60	21.5	29	10	25.8	38	1
girip	3574	5	1	1	2	2	1	1	1	1.2	1.7	1.2	2	25	1.1	12.3	162	50	19.0	23	13	18.9	22	2
datc	1840	5	1	1	2	1	1	2	1	1.2	3.6	1.1	2	27	0.7	11	161	90	32.3	31	12	27.2	21.9	1
rajer	4954	4	1	1	3	3	1	1	1	3.4	2.5	2.1	3	24	1.1	10.2	162	59	22.5	23	8	20.4	13	2
madl	1674	5	1	2	3	1	1	2	1	1.1	4.1	1.8	2	23	0.9	12.4	172	46	15.5	20	7	17.8	23.3	2
muru	6559	3	1	2	3	1	1	1	1	0.8	4.7	1.1	1	18	0.7	12.5	169	84	29.4	34	12	30.2	30.6	1
sivak	7661	5	1	1	2	3	2	1	2	2.3	3.1	1.65	3	19	0.7	8.4	175	72	23.6	27	8	24.4	29	2
vasu	4350	3	1	1	3	2	1	1	1	2.3	3.1	1.3	3	18.4	0.8	7.0	172	70	23.7	24.5	6	22.6	24.2	2
ariva	5584	4	1	1	2	2	1	1	2	3.4	2.1	1.5	1	39	1.2	9.3	169.	55	19.1	25	8	22.4	21.6	2
asait	1165	4	1	1	1	3	1	1	1	13.1	2.4	1.99	3	18	0.7	9.6	168	54	19.1	21	6	19.2	15.3	2
sham	1123	6	1	5	1	2	1	1	2	1.2	3.2	1.4	3	30	1.4	10.3	160	67	26.1	29.5	9	26.6	20.3	2
rama	3986	5	1	1	3	3	1	1	1	5.7	3.1	1.8	3	29	0.6	7.4	178	84	26.5	24	6	22.1	26.7	2
munu	4026	4	1	2	1	3	2	1	1	13	3.3	1.42	3	18	0.6	9.8	160	57.4	22.4	24	8	21.4	22.1	3
bhas	4300	5	1	6	3	2	1	2	1	0.9	3.0	1.7	3	25	0.9	5.8	168	71.1	25.2	26	11	22.5	23	2
sarav	2825	3	1	1	2	3	1	1	2	24.6	3	1.9	3	17	0.8	10.4	159	50.2	19.9	21.5	6	19.6	23	2
chan	2306	5	1	1	4	1	1	1	1	0.3	3.9	1.7	3	24	0.7	6.6	160	53.7	21	24	7	21.8	25.3	2
alia I	4415	4	1	1	1	3	1	1	1	8.6	2.9	1.87	3	18	0.5	9	168	68.1	24.1	23.5	6	21.6	25.8	2
abdu	2598	3	1	1	1	3	2	2	2	3.8	3.0	1.4	4	15	0.5	9.3	163	48.5	18.3	19	5	17.4	18	3
kuma	3455	4	1	1	2	3	2	1	2	14.9	3.3	1.6	3	39	2.2	7.4	162	56.4	21.5	25	6	23.1	27.2	3
jaish	4457	4	1	1	1	3	1	1	1	3.9	3.4	1.44	3	19	0.6	9.5	160	65	25.4	24	6	22.1	31.4	2
irudh	6901	4	1	2	3	1	1	1	1	2.1	3.9	1.09	1	18	1.1	11	163.	60.8	22.7	29	10	25.8	39.8	1
ravi	4844	4	1	1	3	3	1	1	2	4.9	2.8	1.6	3	16	1.0	12.8	162.	40.3	15.2	17	4	15.7	13.9	3
manc	5037	3	1	1	1	3	2	1	2	4.1	2.8	2.23	4	16	0.5	9.4	172	83.1	27.8	26	11	22.5	30.5	2
patc	4921	4	2	6	1	1	1	1	2	0.8	4.4	1.2	3	24	0.5	8.8	148	33.4	15.1	14	4	12.7	8.7	2
gang	7545	4	2	4	3	1	1	1	1	1.4	3.7	1.0	2	26	0.6	7	145	46.1	21.9	20	9	17.1	13.3	2
indra	1497	4	2	1	1	1	1	1	1	0.6	2.8	1.4	1	23	0.7	11	161.	42.7	16.4	18	5	16.4	12.9	2
deva	6912	4	1	1	1	3	1	2	2	2.6	3.4	1.3	3	21	0.6	6.7	166	57.1	20.1	20	8	17.4	24.8	2
kena	5736	6	2	5	1	1	1	1	1	0.7	3.5	1.36	1	34	0.9	8.6	141	51.1	25.9	27	11	23.5	17.5	1
Chin	7360	5	1	1	2	3	2	1	2	8.3	2.6	2.07	3	16	0.5	5.9	165.	55.6	20.3	21	5	19.4	22.1	3
karth	5283	3	1	1	1	2	1	1	1	9.8	3.7	2	2	14	0.5	6.6	171	79.2	27.1	27.5	15	22.7	28.1	2
anan	5182	6	1	1	1	3	2	1	1	3.8	3.0	1.34	3	42	1.6	8.6	166.	66.9	24.1	22.5	6	20.6	20.6	2
Ravi	2183	4	1	1	1	3	1	2	2	7.1	3.2	1.56	2	14	0.5	8.4	153.	44.1	18.7	23	11	19.5	16.9	2
Puru	5389	4	1	1	1	3	2	1	2	4.4	2.7	2.91	3	10	0.6	6.5	161.	83.5	32	27.5	12	23.7	16.6	3
sivak	5970	5	1	1	1	3	1	1	2	24.3	3.0	1.5	3	13	0.9	12.3	164	83.2	30.9	27	11	23.5	18.1	2
Saga	5926	5	1	2	3	1	1	1	1	1.7	4.2	1.1	1	16	0.6	9.3	171.	70.9	24.1	28	16	22.9	32.3	1
abdu	2717	5	1	1	1	3	2	1	1	3.3	2.6	1.7	1	20	0.6	7	156	54	22.2	24	14	19.6	22.9	2
karth	5729	3	1	4	3	3	1	2	1	10.9	2.5	3.1	3	27	0.6	4.8	170	54.4	18.8	17.5	9	14.6	17.8	2
ezhil	5985	5	1	2	1	3	2	1	2	3.4	2.6	1.36	2	24	1.2	11	157	54.8	22.2	22.5	10	19.3	27.5	2

Moor	6331	7	1	1	1	3	1	1	1	9.5	2.3	1.7	2	18	0.6	10	166	80.1	29.1	26.5	10	23.3	25.4	2
Munr	2520	3	1	6	4	1	1	2	1	1.2	4.4	1.2	1	14	0.6	5.3	168	56.6	20.1	23	11	19.5	31.6	1
Wilso	6513	6	1	1	1	3	2	1	1	7.1	2.0	1.9	2	35	0.9	9	161	77	29.7	26.5	12	22.7	24.2	2
Ezhil	35.0	4	1	1	4	2	1	2	2	1.6	4.0	1.65	3	16	0.8	8.9	168	59.8	21.2	22	6	20.1	27.6	2
Seka	6571	6	1	1	1	3	1	2	1	3.2	2.7	1.4	2	17	0.6	7.4	158.	52.3	20.8	22.5	8	19.9	29.4	2
Sent	6727	4	1	2	4	1	1	2	1	1.9	4.0	1.2	1	29	0.2	7.9	155	46	19.1	22.5	10	19.4	29.2	1
mano	5267	6	1	1	1	1	1	1	1	1.5	3.7	1.13	1	28	0.8	11	159	58.4	23.1	25.5	10	22.3	21.4	1
Augu	2864	4	1	1	3	1	1	1	2	1.7	3.3	1.1	1	19	0.6	11.9	173	87.1	29.1	28.5	14	24.1	32.3	1
Raja	5416	7	1	1	1	2	1	2	1	1	2.3	1.43	2	76	1.3	7.8	159	71.6	28.3	30.5	15	25.7	16.5	2
srinivasan		6	1	1	1	3	1	1	1	5.2	2.3	1.13	2	17	0.6	11	173	58.8	19.6	21.5	9	18.6	24.9	2
Gopi	6023	4	1	1	1	3	2	1	2	12.6	2.7	1.68	2	22	0.6	7.4	173	63.3	21.1	23	10	19.8	18.9	3
Slvak	4407	4	1	1	2	2	1	2	1	4.5	3.2	1	1	14	0.7	10.7	168	76.4	27.1	28	12	24.2	34	1
Mani	7239	6	1	1	1	3	1	1	3	13.7	2.5	1.76	2	24	1.0	11	172.	62.5	21	19	8	16.4	20.4	2
Kous	7231	2	2	6	1	2	1	1	1	2.7	3.3	1.02	3	27	0.7	7.0	165	68.8	25.3	23	14	18.6	20.7	1
Sivak	2528	4	1	1	1	3	1	1	1	2.2	2.4	2.3	3	20	0.7	8.4	169	74.3	26	21	10	17.8	22.4	3
Satis	2374	1	1	2	1	3	1	1	1	10.7	2.0	1.54	2	19	0.6	10	164	58.4	21.7	22.5	9	19.6	27.8	2
Venk	590.	4	1	1	2	2	2	2	1	2.9	2.8	1.16	2	75	1.2	6.8	163	44.2	16.6	19.5	6	17.6	24.4	3
subra	7687	6	1	1	1	2	2	2	2	1.0	3.1	1.1	3	77	2.8	6	161	44.2	16.6	20	6	18.1	11	3
Veka	7880	4	1	1	1	2	1	1	1	2.5	3.5	1.0	2	86	2.4	11	164	89.4	33.2	31	13	26.9	46	1
shan	8146	6	1	6	1	2	1	1	1	0.8	3.6	0.9	2	20	0.6	8.6	178	56.5	17.8	20	5	18.4	22.5	2
choc	1021	7	1	1	3	1	1	1	2	0.9	3.2	1.44	2	20	0.5	11.4	147	57.2	26.5	26.5	13	22.4	20.8	2
maha	8422	3	1	1	1	3	2	1	2	3.3	2.2	1.2	2	18	0.8	5.7	161	54.4	21	22.5	12	18.7	20.1	2
kantl	8429	5	1	1	1	3	1	1	1	3.2	2.0	1.2	2	21	0.8	10.2	170	67.9	23.5	25	12	21.2	30.1	2
prisc	6256	7	2	5	2	2	1	1	1	0.8	2.0	1.1	2	50	1.4	8.8	147	65	30.2	25	12	21.2	12.4	2
rajendran		6	1	1	1	3	2	1	1	5.3	2.9	1.3	2	14	0.5	12	160	46.9	18.3	19	8	16.4	15.7	3
krish	14.1	5	1	1	1	3	1	1	1	7.5	2.9	1.2	2	27	0.7	9.5	169	60.8	21.3	22.5	8	19.9	24	2
jana	101.	5	1	1	1	3	2	1	1	4.5	1.6	1.74	3	18	0.8	10	170	76.5	26.5	25	10	21.8	28.5	2
anba	8462	5	1	2	1	3	1	1	2	6.1	3.7	1.8	2	26	0.7	9.0	167	50.2	18	21	8	18.4	22.2	2
vaiya	352.	6	1	1	1	3	2	1	2	5.9	3.7	2.1	3	17	0.9	12.1	162	75.5	28.8	23.5	8	20.9	24.7	3
sathy	225.	5	1	1	1	3	2	2	2	8.8	2.6	1.3	3	18	0.6	6	170	57.3	19.8	20	7	17.8	21.1	3
katha	384.	8	1	2	2	1	1	2	1	0.7	3.3	0.9	1	21	0.7	10.2	167	66.2	23.7	25	15	20.2	22.9	1
selva	632.	1	1	5	2	1	1	2	1	0.8	3.9	1.1	2	21	0.6	6.2	175	67.7	22.1	24.5	12	20.7	25.8	2
logan	5905	5	1	1	3	3	2	2	2	1.7	2.2	1.9	3	57	1.8	8.4	161	44.9	17.3	17.5	6	15.6	14.9	3
rajen	3502	4	1	1	2	2	1	2	1	2.5	1.7	1.2	1	37	1.1	7.4	165	76.3	27.7	32	20	25.7	33.2	1
balu	545.	5	1	1	1	2	1	1	1	8.0	2.8	1.32	1	27	0.7	9.0	168	47.6	16.9	18	6	16.1	16.4	2
selva	814.	5	1	2	2	2	1	2	1	3.0	3.2	1.1	2	19	0.8	10.0	170	68	23.6	25	13	22	28	1

name	ddhd	age	fat	visceralf	ScWB	ScTrunk	ScArm	ScLeg	SkWB	SkTrun	SkArm	Sklegs	REE
raju	7426.12	4	27.9	6.5	18.6	16.3	26.5	26.5	29.7	22.1	37.3	47.1	1373
datchinam	1840.13	5	36.4	22.5	25.6	23.8	35.4	36.3	24.8	16	33	42.4	1729
vasudevan	4350.12	3	22.6	9	15.9	14.3	19.7	19.7	31.6	25	38.2	48.9	1663
ramakrishn	3986.14	5	18.1	8	13.3	12.2	14.9	14.6	36.8	30.3	41.1	54	1882
bhaskar	4300.14	5	26	12	18	16.2	24	24.2	29.7	21.6	35.6	47.1	1598
chandrasek	2306.03	5	23.8	6.5	16	13.9	21.8	21.8	29.2	22.9	36.5	46.6	1322
alia basha	4415.14	4	16.7	8	12	10.8	15	14.5	34.4	28.3	39.8	51.6	1602
abdul ajee	2598.14	3	19	2.5	12.6	10.6	18.6	18.1	33	23	40.5	50.3	1265
kumar	3455.12	4	20.4	6.5	14	12.2	19.3	18.9	31	25.2	37.9	48.3	1382
jaishankar	4457.14	4	19.8	9.5	14.2	12.8	18	17.7	31.6	25.8	38.8	48.9	1527
irudhayana	6901.11	4	16.5	7.5	11.8	10.4	16	15.3	33.3	28	38.6	50.5	1475
ravi	4844.12	4	16.1	0.5	10.3	8.2	15.2	14.5	33.6	28.4	41.5	50.9	1134
manoj	5037.14	3	18.9	9.5	14	12.9	15.6	15.3	35.9	29.3	40.6	53.1	1841
patchaiyan	4921.14	4	21.7	0.5	16.1	12.6	31.2	24	25.3	21.1	32.8	37.8	866
indrani	1497.13	4	33.6	1.0	23	21.4	45	33.9	24.2	20	29.4	32.5	993
deva	6912.13	4	24.1	5	16.1	14	23.1	23	31.1	24.2	38.8	48.5	1386
kenammal	5736.14	6	36.7	9.5	31.3	28	47.9	41.1	20.5	15	23.2	33.7	1098
Chinnappa	7360.13	5	25.9	5.5	17.2	14.9	23.8	23.9	29.6	22.6	37.3	47	1350
karthikeya	5283.14	3	27	11.5	18.9	17.2	26.6	26.7	30.9	23.2	37.2	48.2	1723
anand	5182.14	6	18.5	8	13.2	11.8	16.2	15.8	32.7	27	39.1	50	1568
Ravi	2183.14	4	27.8	3.5	18.1	15.5	25.2	25.6	29.9	22.3	38.2	47.3	1165
Purushotha	5389.14	4	26	13.5	19.1	18	23.1	23.4	30.7	23.3	37.8	48	1802
sivakumar	5970.14	5	32.4	18	22.9	21.2	31.5	32	28.2	18.9	34.9	44.7	1752
Sagayam	5926.14	5	32.6	10	21.9	19.6	31.7	32.2	28.4	19.8	35.8	45.9	1569
abdulla	2717.14	5	30.1	7.5	20.1	17.7	28.3	28.8	28.1	20.3	36.6	45.6	1309
karthik	5729.14	3	26.7	2.5	17.5	15	25.6	25.8	32.6	24.6	40.6	49.9	1344
ezhilarasar	5985.14	5	22.6	7.5	15.4	13.6	21.7	21.5	30.1	23.9	37.6	47.5	1347
Moorthy	6331.14	7	27.4	17.5	19.5	18	23.2	23.7	29	21.7	35.5	46.4	1732
Munna	2520.01	3	17.6	4	12	10.3	18.3	17.6	34.4	28.5	40.5	51.6	1408
Sekar	6571.14	6	16.6	6	11.5	9.9	15.2	14.6	30.7	26.0	37.6	48.6	1321
Senthil	6727.14	4	12.4	3.5	8.6	7.1	13.9	12.7	33.8	29.6	40.2	51.1	1238
manoharar	5267.14	6	23.1	10	16	14.2	20.7	20.7	28.5	22.5	35.6	45.9	1395
Augustine	2864.12	4	28.2	15	20	18.4	26.4	26.6	30.2	32.3	35.9	48.5	1844
Rajagopal	5416.13	7	30.1	18	21.1	19.3	26.7	27.3	26.2	18.8	33.8	43.8	1572
srinivasan	6947.14	6	18	4	12.2	10.4	17	16.4	32.9	27.3	39.4	50.2	1436
Gopinath	6023.14	4	8.9	3.0	6.7	5.7	10.1	8.6	39	34.5	44	56	1567
SIvakumar	4407.13	4	28	12.5	19.6	17.8	27.1	27.3	29.7	22	36.1	47.1	1676
Manimaran	7239.14	6	19.2	3.5	13.1	11.4	17.6	17.2	32.3	26.5	39.6	49.6	1491
Kousalya	7231.14	2			26.4	21.4	38.6	36.4	28.5	23.1	28.8	43.7	1432
Sivakumar2	2528.14	4	18.8	6.5	13.7	12.5	17.2	16.7	34.4	28.2	40.7	51.6	1700
Satish	2374.14	1	14.1	5.5	10.2	8.9	15	14	34.5	29.7	41	51.9	1447
Venkatesar	590.13	4	19.4	0.5	12.6	10.4	18.3	17.9	33.2	27.1	41	50.4	1193
Vekatesan	7880.14	4	23.8	18	17.9	17	18.8	19.1	33.7	26.3	37.9	50.9	1929
shanavas	8146.14	6	8.6	1.0	6.0	6.0	9.5	8.0	37.1	33.2	42.4	54.2	1440
chockkalin	1021.10	7	38.1	15.5	25.7	23.2	35.3	36.5	22.3	13.6	37.7	39.9	1313
kantharaj	8429.14	5	21.6	8.0	15	13.3	20.5	20.2	32.2	25.8	38.7	49.5	1573
priscilla	6256.13	7	36	9.5	34.3	29.7	45.8	41	23.4	16.6	23.6	39	1376

rajendran	7222.1	6	46.9	19.9	13.1	11.1	17.7	17.4	29.5	24.2	37.4	46.9	1222
krishnamoo	14.15	5	16.8	5.5	11.7	10.1	16.4	15.7	33.8	28.3	40	51	1477
janardhana	101.15	5	20.6	10	14.9	13.6	18	17.6	33.4	26.4	34.3	50.6	1724
anbalagan	8462.14	5	16.2	2.0	10.8	8.9	15.4	15.2	33.4	28.2	40.1	50.6	1300
vaiyapuri	352.15	6	24.1	14.5	17.4	16.1	20.4	20.6	30.5	23.7	37.2	47.8	1678
sathyamoo	225.15	5	15.4	3.5	10.6	9.0	15.7	14.8	34.5	29.2	40.8	51.7	1425
kathavaray	384.15	8	39.3	11.5	26	23.2	36.1	37.4	24.5	14.9	33	42.1	1455
selvaraj	632.15	1	21.1	7.5	14.5	12.7	19.1	18.9	32.1	25.8	37.6	49.4	1372
rajendran	3502.14	4	24.3	13	17.3	15.9	22.5	22.5	31.2	24.2	36.7	48.4	1696
balu	545.15	5	20.5	1.0	13.3	11	18.6	18.4	32.1	26	39.8	49.4	1243
selvaraj	814.15	5	36	8	16	14	20.4	20.4	31	26	40	47	1580